



Steele, James. (2015). Isolated lumbar extension exercise as an intervention for chronic low back pain. PhD thesis, Southampton Solent University.

Downloaded from <http://ssudl.solent.ac.uk/3173/>

Usage Guidelines

Please refer to usage guidelines at <http://ssudl.solent.ac.uk/policies.html> or alternatively contact ir.admin@solent.ac.uk.

**Isolated Lumbar Extension Exercise as an
Intervention for Chronic Low Back Pain**

James Daniel Steele

Centre for Health, Exercise & Sport Science, Southampton Solent
University, Southampton, Hampshire, UK

**A thesis submitted in partial fulfilment of the requirements of Nottingham Trent
University and Southampton Solent University for the degree of Doctor of
Philosophy**

March 2014

Copyright Statement

This work is the intellectual property of James Steele. You may copy up to 5% of this work for private study, or personal, non-commercial research. Any re-use of the information contained within this document should be fully referenced, quoting the author, title, university, degree level and pagination. Queries or requests for any other use, or if a more substantial copy is required, should be directed in the owner(s) of the Intellectual Property Rights.

ABSTRACT

Low back pain (LBP) is highly prevalent, generally categorised as 'non-specific' as clear diagnosis for pain is often absent, and further categorised into acute, sub-acute and chronic, with 69-75% of acute cases developing into chronic. This chronic LBP population accounts for the majority of economic costs worldwide associated with LBP. Although LBP is often 'non-specific', many physical dysfunctions are associated with it. Thus LBP can be regarded as multifactorial in nature. Dysfunctions include, but are not limited to: deconditioning of the lumbar extensor musculature, limited range of motion (ROM), gait abnormality and disc disorders. The novel approach of this thesis was to consider lumbar extensor deconditioning, LBP and its associated physical dysfunctions within a multifactorial framework, and the potential improvement of associated dysfunctions from intervention using isolated lumbar extension (ILEX) specifically aimed at addressing lumbar extensor deconditioning. Findings from three empirical studies are reported. The first examined limited ROM ILEX exercise compared with full ROM exercise. Results from this study support that limited ROM training is as effective as full ROM training at improving full ROM ILEX strength, pain and disability. The second study examined the effects of ILEX exercise upon lumbar spine kinematic waveform pattern variability during gait. Results from this study demonstrate that ILEX exercise significantly improves sagittal plane variability in chronic LBP participants. The final study examined the effects of ILEX exercise upon disc hydration determined indirectly through measurement of spinal height using seated stadiometry. Results from this study showed improved ILEX strength, pain and disability but did not demonstrate improvement in disc hydration. These results provide evidence for adopting a multifactorial conceptualisation of LBP in the use of ILEX exercise as a treatment. It is concluded that a wide range of improvements including pain, disability and various aspects of function relating to the multifactorial model are possible through use of a single minimal intervention involving ILEX. This conclusion has potential implications for considering direction of treatments from clinicians towards chronic LBP. Such a minimal intervention offering a wide range of benefits may reduce the need for costly and complex multi-disciplinary interventions.

Acknowledgements

Firstly I'd like to take the time to thank my wonderful girlfriend and love of my life Emma. Without her providing the initial impetus all those years ago to break out of my boring job and choose to study something I was passionate about I'm not sure I'd be here writing this today. Her patience and support while I have been a student for what seemed like forever has been immeasurable. Don't worry, now I can go get a real job and start taking care of you!!

I'd like to thank the late Arthur Jones for his contributions to this area through both Nautilus and MedX as in all likelihood again I probably wouldn't be here studying this particular area if it were not for his efforts.

My supervisors deserve particular thanks also. Dr Stewart Bruce-Low has maintained a constant supportive role through both the highs and the lows of this process. He has invested far more time to in this process than his responsibility entailed. Hopefully my accomplishment will be reward for his effort – oh, that and the awesome trip to Dubai ;-) – Dr Dave Smith receives my thanks for remaining down to earth through this whole process and keeping me grounded – that and being the most efficient 'Grammar Nazi' I've ever met. He made me realise that even during the PhD there is time to enjoy yourself and relax. Dr David Jessop is a biomechanical wizard, and he's 'dead good' on MatLab also. He often pointed me in the right direction after hours of frustration.

Support from my family has been ever present through this process. My Mum and Dads constant questioning of *'When are you going to finish?...So when can we call you doctor?'* along with grandparents constant bragging of my achievements to their friends spurred me on to get finished sooner rather than later so they could relish in the final achievement with me. My younger brothers get a thank you also for not being too much of a pain throughout and for being surprisingly insightful (yes, all of them) when discussing my work with them.

My friends have been there throughout the whole process too. Sean – ‘*Me and him are....buddies*’ – for being my best friend and constant inspiration through his own achievements. James for being my friend, colleague and official coffee time partner, providing laughs and serious discussion on the world in its entirety. The guys who have enabled me to keep up my hobbies in roleplaying and wargaming throughout the process – Ross, Lea, Adam, Ian, Dave, Simon, John, Ant – that escapism has been much needed.

I’d like to thank the staff here at Solent and everyone else who helped out with the work here particularly Tim, Grant, and James for accommodating my perpetual presence in the labs, Scott for his insights on research methods, and Dr Neil Osborne for his efforts in screening participants and providing critical feedback on manuscripts.

Finally I’d also like to thank the research community at Solent, including Profs, supervisors, support staff and the PGR students themselves, for being so welcoming and really working to foster and encourage a friendly and constructive research environment here.

CONTENTS

<u>Section</u>	<u>Pg. No.</u>
ABSTRACT	iii
Acknowledgements	iv
List of Tables	xiii
List of Figures	xiv
Glossary	xvi
1.0 INTRODUCTION	1
1.1 Defining Low Back Pain within a Multifactorial Model	1
1.2 Prevalence, Costs and Impact	3
1.3 Lumbar Spine Anatomy	6
1.4 Mechanisms of Pain within LBP	8
1.5 A Multifactorial Perspective of Low Back Pain	14
2.0 LITERATURE REVIEW	17
2.1 Introduction and Overview	17
2.2 A Reappraisal of the Deconditioning Hypothesis in Low Back Pain: Review of Evidence from a Triumvirate of Research Methods on Specific Lumbar Extensor Deconditioning	19
2.2.1 Overview	19
2.2.2 Introduction	20
2.2.2.1 Defining the Disuse/Deconditioning Hypothesis – ‘Disuse’ OR ‘Deconditioning’?	20
2.2.2.2 Specific Disuse/Deconditioning of the Lumbar Extensors	24
2.2.2.3 Aim and Approach of this Review	24

2.2.3 Strength and Endurance of the Lumbar Extensor Musculature in LBP	26
2.2.3.1 Considerations for Studies of Strength and Endurance of the Lumbar Extensor Musculature	26
2.2.3.2 Isolated Lumbar Extension Studies	29
2.2.3.3 Summary of Strength and Endurance Studies of the Lumbar Extensor Musculature	31
2.2.4 Imaging and Histochemical Studies of the Lumbar Extensor Musculature in LBP	34
2.2.4.1 Considerations for Imaging and Histochemical Studies of the Lumbar Extensor Musculature	34
2.2.4.2 Imaging Studies of the Lumbar Musculature	36
2.2.4.3 Histochemical Studies of the Lumbar Musculature	40
2.2.4.4 Summary of Imaging and Histochemical Studies of the Lumbar Extensor Musculature	42
2.2.5 Electromyography Studies of Fatigue in the Lumbar Extensor Musculature in LBP	46
2.2.5.1 Considerations for Electromyographic Fatigue Analysis of the Lumbar Extensor Musculature	46
2.2.5.2 Fatigability Studies of the Lumbar Musculature	49
2.2.5.3 Summary of Electromyographic Studies of Fatigue of the Lumbar Extensor Musculature	52
2.2.6 Prospective Studies of Lumbar Extensor Deconditioning in LBP	54
2.2.6.1 Prospective Evidence from Strength and Endurance in LBP	55
2.2.6.2 Prospective Evidence from MRI & EMG in LBP	58
2.2.6.3 Summary of Prospective Studies	59
2.2.7 Discussion	67
2.2.8 Conclusion	71
2.3 A Review of the Specificity of Exercises Designed for Conditioning the Lumbar Extensors	72

2.3.1 Overview	72
2.3.2 Introduction	73
2.3.3 Methods	75
2.3.3.1 Exercises Designed for the Lumbar Extensors	75
2.3.3.2 Validity of Outcome Measures	76
2.3.4 Results	79
2.3.4.1 Acute Activation of the Lumbar Extensors through Specific Exercise	79
2.3.4.2 Chronic Adaptation to Specific Exercise Training for the Lumbar Extensors	86
2.3.5 Discussion	91
2.4 Clinical use of Specific Exercise for the Isolated Lumbar Extensors in Chronic Low Back Pain	96
2.4.1 Overview	96
2.4.2 Introduction	97
2.4.3 Methods	101
2.4.4 Results	101
2.4.4.1 Pain, Disability and Clinical Meaningfulness of Outcomes	116
2.4.4.2 Manipulation of Resistance Training Variables for use of ILEX	122
2.4.4.3 Studies of ILEX and other Specific Exercise Approaches	125
2.4.5 Discussion	128
2.4.6 Conclusion	133
2.5 Synthesis and Overarching Rationale for the Thesis	134
2.6 Areas of Empirical Study	136
2.6.1 Range of Motion and Isolated Lumbar Extension Exercise in Chronic Low Back Pain	136
2.6.2 Gait Variability and Isolated Lumbar Extension Exercise in Chronic Low Back Pain	138

2.6.3 Intervertebral Disc Hydration and Isolated Lumbar Extension Exercise in Chronic Low Back Pain	140
2.7 Summary of Literature Review	143
2.8 Proposed Research Questions and Hypotheses	144
3.0 METHODS	145
3.1 Study Design	145
3.1.1 Study Design; Study 1 and Study 2	145
3.1.2 Study Design; Study 3	145
3.2 Participants	146
3.2.1 Participants; Study 1 and Study 2	147
3.2.2 Participants; Study 3	149
3.3 Equipment	150
3.3.1 Equipment; Study 1 and Study 2	150
3.3.2 Equipment; Study 3	151
3.4 Participant Testing	153
3.4.1 Participant testing; Study 1 and Study 2	153
3.4.1.1 Three Dimensional Motion Analyses	153
3.4.1.2 Biomechanical Model	154
3.4.1.3 Marker Set Up	154
3.4.1.4 Kinematic Data	155
3.4.2 Participant Testing: Study 3	158
3.5 Participant Training	160
3.6 Data Analysis	161
3.5.1 Data Analysis; Study 1	161
3.5.2 Data Analysis; Study 2	162
3.5.3 Data Analysis; Study 3	162

4.0 RESULTS & DISCUSSION	164
4.1 Isolated Lumbar Extension Exercise in Chronic LBP: Comparison of Limited Range of Motion and Full Range of Motion Isolated Lumbar Extension Exercise	164
4.1.1 Results	164
4.1.1.1 Participants	164
4.1.1.2 Isolated Lumbar Extension Strength	165
4.1.1.3 Lumbar and Standing Range of Motion	166
4.1.1.4 Pain and Disability	166
4.1.2 Discussion	167
4.2 Isolated Lumbar Extension Resistance Exercise Effects Upon Gait Variability in Chronic Low Back Pain Participants	173
4.2.1 Results	173
4.2.1.1 Participants	173
4.2.1.2 Baseline Kinematic Data	173
4.2.1.3 Effects of Intervention upon Kinematic Variables	176
4.2.2 Discussion	178
4.3 The Effects of Isolated Lumbar Extension Resistance Training Upon Disc Hydration Measured Indirectly through Seated Stadiometry in Participants with Chronic Low Back Pain	110
4.3.1 Results	184
4.3.1.1 Participants	184
4.3.1.2 Seated Stadiometry	184
4.3.1.3 Isolated Lumbar Extension Strength	185
4.3.1.4 Pain and Disability	185
4.3.2 Discussion	186
5.0 CONCLUSIONS	191
5.1 Future Directions	195
5.1.1 Study 1	195

5.1.2 Study 2	197
5.1.3 Study 3	198
5.1.4 General	199
6.0 REFERENCES	202
7.0 APPENDICES	262
7.1 Published Work	
7.1.1 Peer Reviewed Journal Articles	
7.1.2 Conference Presentations	
7.2 Top Lesson(s) Learnt from Undertaking the Ph.D	
7.3 Ethics Documentation	
7.3.1 Southampton Solent University Ethics Approval – Study 1 & 2	
7.3.2 NHS Research Ethics Service Approval – Study 1 & 2	
7.3.3 Southampton Solent University Ethics Approval – Study 3	
7.4 Participant Documentation	
7.3.1 Study 1 & 2 Participant Information Sheet	
7.3.2 Study 1 & 2 Informed Consent Form	
7.3.3 Study 3 Participant Information Sheet	
7.3.4 Study 3 Informed Consent Form	
7.3.5 Participant Referral Form	
7.5 MedX Equipment Operation Guidelines	
7.6 Summary Tables from Deconditioning Review	
7.5.1 Summary of studies testing strength and endurance of the lumbar extensor musculature in LBP	
7.5.2 Summary of imaging and histochemical studies of the lumbar extensor musculature in LBP	
7.5.3 Summary of studies testing fatigability with EMG of the lumbar extensor musculature in LBP	

7.5.4 Summary of prospective studies of lumbar extensor musculature
deconditioning in LBP

7.7 Determining the reliability of a custom built seated stadiometry set-up for
measuring spinal height in participants with chronic low back pain

LIST OF TABLES

<u>Table</u>	<u>Pg. No.</u>
Table 1. Summary of studies testing strength and endurance of the lumbar extensor musculature in LBP	32
Table 2. Summary of imaging and histochemical studies of the lumbar extensor musculature in LBP	43
Table 3. Summary of studies testing fatigability with EMG of the lumbar extensor musculature in LBP	53
Table 4. Summary of prospective studies of lumbar extensor musculature deconditioning in LBP	61
Table 5. Summary of studies examining ILEX in CLBP upon pain, disability and GPOs	120
Table 6. Participant Baseline Demographics (Study 1)	164
Table 7. Change in lumbar and standing ROM	166
Table 8. Change in VAS and ODI (Study 1)	167
Table 9. Participant Baseline Demographics (study 2)	173
Table 10. Pre and Post Kinematic data	177
Table 11. Participant Baseline Demographics (Study 3)	184
Table 12. Seated stadiometry result from each time point.	184
Table 13. Change in VAS and ODI (Study 3)	186

LIST OF FIGURES

<u>Figure</u>	<u>Pg. No.</u>
Figure 1. Integrated multifactorial framework of function (Lee & Vleeming, 1998)	15
Figure 2. Disuse Syndrome Model, from Verbunt et al. (2003)	20
Figure 3. Deconditioning Syndrome Model – Adapted* Disuse Syndrome Model, adapted from Verbunt et al. (2003)	23
Figure 4. Example of a restraint system used to allow isolated lumbar extension (ILEX) exercise and testing to be performed (Reprinted with permission from MedX Corporation)	99
Figure 5. Power Analysis to determine participant numbers	146
Figure 6. CONSORT diagram to illustrate participant numbers for enrolment, allocation, follow-up and analysis stages for study 1	148
Figure 7. CONSORT diagram to illustrate participant numbers for enrolment, allocation, follow-up and analysis stages for study 2	149
Figure 8. Flow of participants through study 3	150
Figure 9. Custom built wall mounted adjustable postural rods.	152
Figure 10. Schematic of seated stadiometry setup.	152
Figure 11. Three dimensional motion camera set up	154
Figure 12. Marker arrangement	155
Figure 13. Example of limited ROM using the mid 50% of participants individual full ROM, in this case 72°.	160
Figure 14. Pre and post mean isometric ILEX strength curves plotted by quartile across the ROM.	165
Figure 15. Waveform patterns, Winters CV, CV _p and CV _o	175
Figure 16. Example participant data showing pre and post individual trial waveforms and pre and post waveform variability transformed to zero (CV _p)	177
Figure 17. ILEX strength at each time point. Note; *indicates significant	185

pairwise comparison between both T1 and T3, and T2 with T3

GLOSSARY¹

Throughout the thesis when '**the author**' is used it refers to James Steele, the author of the thesis

Important terms and acronyms highlighted here are in alphabetical order

- **1RM** – One repetition maximum
- **BMD** – Bone mineral density
- **BMI** – Body mass index
- **CBT** – Cognitive behavioural therapy
- **Chronic LBP (CLBP)** – LBP having lasted for >12 weeks
- **CNS** – Central nervous system
- **CSA** – Cross sectional area
- **CT** – Computed tomography
- **CV_o** - Variability of the mean offset in ensemble average of a time-series waveform
- **CV_p** – Variability of the pattern in ensemble average of a time-series waveform
- **Deconditioning** – Decrease in function
- **Disc Degeneration** – A physiologic process associated with aging. More severe degeneration and/or structural abnormality may be indicative of a pathological process or injury and is commonly present in those suffering from chronic LBP
- **Disuse** – Decrease in physical activity levels
- **EMG** – Electromyography
- **EMG fatigue indices** - Methods of analysing the EMG signal for determination of fatigability e.g. root mean square amplitude, mean, median or mode frequency slopes, initial frequencies etc.
- **ES** – Erector spinae
- **Gait Variability** – Abnormal variation in gait parameters
- **GPO** – Global perceived outcome

¹ Two copies of the glossary are provided with one for removal by reader to use as reference and bookmark whilst reading the thesis.

- **Isolated Lumbar Extension (ILEX)** – Lumbar extension performed when the pelvis has been appropriately restrained to prevent movement
- **LBP** – Low back pain
- **Lumbar Extensor Musculature** – Lumbar erector spinae and multifidus
- **MCIC** – Minimal clinically important change
- **MF** - Multifidus
- **MMF** – Momentary muscular failure
- **MOS** – Medical outcomes study
- **MRI** – Magnetic resonance imaging
- **Multifactorial Framework** - An integrated multifactorial framework of function considering all potentially interrelated deficiencies in function in LBP under the areas of; form closure (structure), force closure (force produced by myofascial action), motor control (neural patterning and control), and emotions (psychological and psycho-social factors)
- **MVC** – Maximal voluntary contraction
- **Non-specific LBP** – LBP for which it is not possible to identify a specific cause of pain
- **ODI** – Oswestry disability index
- **PNS** – Peripheral nervous system
- **QL** – Quadratus lumborum
- **Resistance Training** – An exercise modality performed with the goal of conditioning the muscles (i.e. increasing strength, endurance or hypertrophy) using external resistance (i.e. free weights, bodyweight exercise, variable resistance machines, hydraulic resistance machines, and pneumatic resistance machines)
- **RMDQ** – Roland Morris disability questionnaire
- **ROM** – Range of motion
- **Seated Stadiometry** – Measurement of seated stature for indirect determination of spinal height and disc hydration
- **SF36** – Short form 36 health questionnaire

- **Stad1st** – The first measurement taken during each seated stadiometry trial
- **StadAvg** – The average of the ten measurements taken during each seated stadiometry trial
- **StadShrink** – The difference between the first and last of the 10 measurements taken during each seated stadiometry trial
- **Trunk Extension (TEX)** – A compound movement involving both hip extension and lumbar extension
- **TSK** – Tampa scale for kinesiophobia
- **VAS** – Visual analogue pain scale
- **Winters CV** – Method typically used to calculate the variability in ensemble average of a time-series waveform

GLOSSARY

Throughout the thesis when '**the author**' is used it refers to James Steele, the author of the thesis

Important terms and acronyms highlighted here are in alphabetical order

- **1RM** – One repetition maximum
- **BMD** – Bone mineral density
- **BMI** – Body mass index
- **CBT** – Cognitive behavioural therapy
- **Chronic LBP (CLBP)** – LBP having lasted for >12 weeks
- **CNS** – Central nervous system
- **CSA** – Cross sectional area
- **CT** – Computed tomography
- **CV_o** - Variability of the mean offset in ensemble average of a time-series waveform
- **CV_p** – Variability of the pattern in ensemble average of a time-series waveform
- **Deconditioning** – Decrease in function
- **Disc Degeneration** – A physiologic process associated with aging. More severe degeneration and/or structural abnormality may be indicative of a pathological process or injury and is commonly present in those suffering from chronic LBP
- **Disuse** – Decrease in physical activity levels
- **EMG** – Electromyography
- **EMG fatigue indices** - Methods of analysing the EMG signal for determination of fatigability e.g. root mean square amplitude, mean, median or mode frequency slopes, initial frequencies etc.
- **ES** – Erector spinae
- **Gait Variability** – Abnormal variation in gait parameters
- **GPO** – Global perceived outcome
- **Isolated Lumbar Extension (ILEX)** – Lumbar extension performed when the pelvis has been appropriately restrained to prevent movement

- **LBP** – Low back pain
- **Lumbar Extensor Musculature** – Lumbar erector spinae and multifidus
- **MCIC** – Minimal clinically important change
- **MF** - Multifidus
- **MMF** – Momentary muscular failure
- **MOS** – Medical outcomes study
- **MRI** – Magnetic resonance imaging
- **Multifactorial Framework** - An integrated multifactorial framework of function considering all potentially interrelated deficiencies in function in LBP under the areas of; form closure (structure), force closure (force produced by myofascial action), motor control (neural patterning and control), and emotions (psychological and psycho-social factors)
- **MVC** – Maximal voluntary contraction
- **Non-specific LBP** – LBP for which it is not possible to identify a specific cause of pain
- **ODI** – Oswestry disability index
- **PNS** – Peripheral nervous system
- **QL** – Quadratus lumborum
- **Resistance Training** – An exercise modality performed with the goal of conditioning the muscles (i.e. increasing strength, endurance or hypertrophy) using external resistance (i.e. free weights, bodyweight exercise, variable resistance machines, hydraulic resistance machines, and pneumatic resistance machines)
- **RMDQ** – Roland Morris disability questionnaire
- **ROM** – Range of motion
- **Seated Stadiometry** – Measurement of seated stature for indirect determination of spinal height and disc hydration
- **SF36** – Short form 36 health questionnaire
- **Stad1st** – The first measurement taken during each seated stadiometry trial

- **StadAvg** – The average of the ten measurements taken during each seated stadiometry trial
- **StadShrink** – The difference between the first and last of the 10 measurements taken during each seated stadiometry trial
- **Trunk Extension (TEX)** – A compound movement involving both hip extension and lumbar extension
- **TSK** – Tampa scale for kinesiophobia
- **VAS** – Visual analogue pain scale
- **Winters CV** – Method typically used to calculate the variability in ensemble average of a time-series waveform

1. INTRODUCTION

1.1 Defining Low Back Pain

Low back pain (LBP) is pain localized to the lumbar area ranging from the inferior ribcage to the waistline (12th thoracic/1st lumbar to 5th lumbar/1st sacral vertebrae) and often includes radiating leg pain such as sciatica. It is often labelled ‘non-specific’¹ and has been described by the National Institute for Health and Clinical Excellence (NICE, 2009) as “...*tension, soreness and/or stiffness in the lower back region for which it is not possible to identify a specific cause of the pain*” (pp. 4). It is estimated that in as much as 85% of LBP cases no specific patho-anatomical diagnosis can be found (White & Gordon, 1982; Nachemson et al., 2000). Non-specific LBP is further categorised as acute, sub-acute or chronic. Acute pain occurs suddenly and lasts <6 weeks, sub-acute pain lasts between 6-12 weeks, whereas chronic pain develops gradually, lasting >12 weeks and is often recurring (Frymoyer, 1988). The ‘natural history’ of LBP is described as the majority of low back injuries and acute LBP cases recovering before chronicity develops, and the enormous costs associated with LBP are due to the sub-group of those with chronic LBP² (NICE, 2009). Thus chronic LBP is looked upon by some as an entirely different entity from low back injury and acute LBP (Balague et al., 2012). However, while there are a variety of further co-morbidities associated with, and which develop with, chronic LBP, such as psycho-social factors, it should be noted that logically all “...*chronic back pain always starts as acute back pain*” (Adams et al., 2010, pp. 967). Indeed, in contrast to the common notion of LBP’s natural history involving recovery of most acute LBP, evidence suggests that a considerable proportion (69% to 75%) of low back injury and acute LBP develops into chronic LBP (Papageorgiou et al., 1996; Croft et al., 1998), often with increasing frequency and severity (Donelson et al., 2012). Though most people visiting

¹ As will be noted in a later section of this introduction, there are a number of possible sources of pain causing mechanisms in LBP although it is often difficult to ascribe in individual cases using diagnostic techniques what is specifically causing pain. This is one of the reasons for a high proportion of LBP being labelled ‘non-specific’ i.e. there is *absence of evidence* (not meaning *evidence of absence*) for a specific pain mechanism. However, a small proportion of LBP (1%-4%) is identified as ‘Red Flag LBP’ including serious causes of pain such as tumour or malignancy, cauda equina syndrome, infection, or spinal fracture (Downie et al., 2013). These types of LBP are beyond the scope of this thesis to consider.

² Throughout this thesis low back pain ‘in general’ as a non-specific multifactorial condition is discussed (as the majority of all acute low back pain and low back injury develops into a chronic condition [Papageorgiou et al., 1996; Croft et al., 1998] and indeed logically ‘*chronic back pain always starts as acute back pain*’ [Adams et al., 2010, pp. 967]), whereas when discussing specific sub-groups of LBP categorised by symptom duration based upon Frymoyer’s (1988) classifications (for example when discussing the specific population sampled in a cited study or the empirical work conducted towards this thesis), it is clarified as either acute LBP, sub-acute LBP or chronic LBP in the instance it is used.

their physician for LBP complaints will cease to continue consulting them after one month, many continue to experience pain one year after (Papageorgiou et al., 1996; Croft et al., 1998) which suggests patient dissatisfaction with lack of improvement from the care received. This also reinforces that many who suffer from an acute low back injury or acute LBP will likely go on to have persistent chronic symptoms.

Chronic LBP is a multifactorial condition with a wide variety of associated physical dysfunctions including but not limited to; limited lumbar range of motion (ROM; Holmes et al., 1996; Nelson et al., 1995), gait abnormality (Lamoth et al., 2004; Anders et al., 2005; Lamoth et al 2006^a; Lamoth et al 2006^b; Lamoth et al., 2008; Carpes et al., 2008; Tsao & Hodges, 2008; Papadakis et al., 2009; Da Fonseca et al., 2009), and degenerated intervertebral discs (Adams & Roughley, 2006; Maurer et al. 2011; Endean et al., 2011; McNee et al., 2011; Shambrook et al., 2011). The National Research Council (NRC) and The Institute of Medicine (IOM) expert panels acknowledge this and the multifactorial nature of musculoskeletal disorders in the population as a whole (NRC, 1998; NRC & IOM, 2001). Indeed in recent years a number of models attempting to explain, predict and integrate the multifactorial nature of LBP have emerged within the literature (Langevin & Sherman, 2007; Richmond, 2012; Hodges & Smeets, 2014). Although a range of symptoms and dysfunctions may present in chronic LBP it is not always clear whether they are directly responsible for pain experienced, and indeed there are many possible pain causing mechanisms (discussed below) that might be related to these dysfunctions. LBP continues to present as a highly prevalent and costly condition despite continued research into its causes and treatments.

1.2 Prevalence, Costs and Impact

“Back pain is one of the most costly conditions for which an economic analysis has been carried out in the UK”

(Maniadakis & Gray, 2000, pp. 95).

In the UK, direct health care costs of LBP were £1632 million in 1998 (Maniadakis & Gray, 2000). There is also an associated economic loss due to the cost of informal care and production losses (Smith et al., 2008). In the UK, up to 50 million working days are lost each year as a result of LBP (Waddell et al., 2002). This brings the estimated total economic cost of LBP to between £5 billion and £10 billion (Maniadakis & Gray, 2000; Waddell et al., 2002). The majority of these on-going costs come from those suffering from chronic LBP (NICE, 2009). These costs exist for the majority of the western industrialised societies (Smith et al., 2008). A cost of illness study of LBP in the Netherlands during 1991 revealed total costs constituted 1.7% of Gross National Product (Van Tulder et al., 1995). This totalled \$4.6 billion of which 93% accounted for indirect costs such as absenteeism and disablement. LBP was also the most expensive disease category regarding work losses. The total economic cost during 2001 in Sweden was estimated at 1.86 billion Euros; 84% of this accounted for through indirect costs (Ekman et al., 2005). In the US during the 1980s estimates suggest the cost was billions of dollars annually (Pollock et al., 1989) and it seems in some areas prevalence of LBP has been rising since that time (+6.3%), as well as subsequent use of the health care system (+10.9%; Freburger et al., 2009). US reports show that LBP is a common cause of lost work days potentially contributing considerable indirect economic costs (Stewart et al., 2003; Ricci et al., 2006). An estimated 149 million working days are lost each year due to LBP (Guo et al., 1999) which (if indirect costs from the UK and Sweden are comparable) likely represents a huge indirect cost to the US economy. Total costs to the US economy have been recently estimated as high as \$100-200 billion annually (Katz, 2006). Freburger et al., (2009) also reported the number of people utilising government health insurance

(i.e. Medicare and Medicaid) has increased significantly suggesting the majority of increased direct costs from health care utilisation have been supported through tax funded systems. Though different health and social care systems are not directly comparable (Dagenais et al., 2008) it is evident that the prevalence of LBP is a major contributor to costs placed upon the health services in western civilised societies.

Typical treatments used for LBP contribute to the high direct costs associated with LBP (Katz, 2006). For example, Katz (2006) highlighted that General Practitioner (GP) and Physician visits are estimated to cost ~£100 each increasing to around ~£6000 for medical admissions. Surgery costs significantly more ranging from ~£21000 to ~£55500, yet, surgery can often be avoided through more cost effective means (as will be highlighted below). Van Tulder et al. (1995) estimated that total direct medical costs from treatment constituted \$367 million of which \$200 million was accounted for by hospital care costs. This is likely due to the higher costs of hospital care (\$3856; ~£5782; per inpatient). However, although outpatient care is relatively cheaper (\$199; ~£298; per outpatient), the high rates of LBP incidence to be highlighted mean this adds up considerably.

According to the Office of National Statistics (ONS) Omnibus Survey (presented in the Social Trends report) 40% of adults in the UK had suffered from back pain lasting for more than one day in the previous twelve months (ONS, 2000). Evidence for prevalence does vary at a given point in time, as well as duration, and the wide range of figures between studies has been ascribed to lack of uniformity in methods (12-33% for point prevalence, 22-65% for 1 year prevalence, 11-84% lifetime prevalence; Walker, 2000). More recently it has been suggested that one third of the UK population are affected each year (NICE, 2009) and Waddell and Burton (2000) estimate 60 – 80 % of adults will suffer from LBP at some point in their life. Andersson (1999) presented numerous studies further demonstrating high rates of LBP prevalence, including chronic LBP and those with recurrent LBP. The ONS Social Trends (ONS, 2000, p. 122, table. 7.13; ONS, 2010, p.

102, table. 7.16) reports for 1998 and 2008 highlight similar rates of LBP over these ten years. However, the 2000 report illustrates that the ten years preceding it (1988-1998) saw a sharp rise in sickness and invalidity benefit for LBP. In Contrast, US estimates of claims due to LBP decreased by 34% between 1987 and 1995 (Borenstein, 2000) but there is no indication of whether prevalence changed at all. The data of Freburger et al., (2009), however, show that US prevalence may have increased slightly (~6.3%). Whether this truly reflects increased prevalence of LBP, or merely less willingness to tolerate, it is unclear (ONS, 2000). It is difficult to confirm either explanations through observational data on prevalence, though the ONS Health of Adult Britain survey, whilst echoing data on increased sickness and invalidity benefit claims, presented data showing that prevalence barely changed between 1971 and 1981 (ONS, 1997).

Despite most studies utilising differing methods, it is apparent that LBP is highly prevalent (and has been so for some time), and costly in western civilised populations. The World Health Organisation (WHO) also reported rates of back disorders as being highly ranked worldwide as a cause of morbidity (WHO, 1998). Indeed even non westernised indigenous populations are afflicted with comparably high rates of LBP; including Australian Aborigines (Honeyman & Jacobs, 1996; Vindigni et al., 2004; Vindigni et al., 2005; Steering Committee for the Review of Government Service Provision, 2007), Finnish and Sami reindeer herders (~35-60%; Nayha et al., 1991; Daerger et al. 2003) and rural Chinese (64%; Barrero et al., 2006). It is interesting that there is high prevalence of LBP in indigenous populations yet rates of other so called '*diseases of civilisation*' such as obesity, cancer, heart disease, type II diabetes etc., (which have risen dramatically in prevalence over the previous half century in western populations) are almost non-existent when adhering to their traditional diet and lifestyles (Lindeberg et al., 2003; Price, 2008; Carrera-Bastos et al., 2011). Potential evidence of LBP in early hominids (Bonmati et al., 2010), reports of lower back disorders in ancient Egyptian and Nubian remains (Bourke, 1971), as well as an evolutionary basis for lumbar spinal deficiencies contributing to LBP (Lovejoy, 2007; Filler, 2007; Steele, 2013^a) suggests that *Homo sapiens* may even be

anatomically predisposed to LBP, potentially explaining its high prevalence across a range of populations. This wide-spread prevalence across culture gaps suggests that a physical factor, independent of cultural and psychosocial influences, may be predominantly implicated in the multifactorial nature of LBP.

It is apparent that, despite difficulty accurately identifying true prevalence due to different methods, most studies highlight LBP as an issue extending across a wide range of populations. The individual burden, as well as economic costs that such rates of prevalence present is clear and as such understanding LBPs etiology as well as identifying effective treatments is vital to reducing this.

1.3 Lumbar Spine Anatomy

The anatomy of the lumbar spine is complex with bony elements consisting of the vertebrae, the intervertebral discs between the vertebral bodies, the ligaments reinforcing and passively supporting the vertebrae as well as the musculature actively supporting and providing movement, and finally the spinal cord and nerves innervating the local musculature (Drake et al., 2008). From a evolutionary comparative anatomy perspective the present lumbar morphology of *Homo sapiens* represents a gross structure encompassing a wide and short pelvis, long flexible lumbar column and both comparatively large hip extensors (gluteal and hamstring musculature) and small lumbar extensors (erector spinae and multifidus; Lovejoy, 2007; Steele, 2013^a).

The lumbar spine consists of the 5 vertebrae from L1 to L5 and encompassing the L5-S1 lumbopelvic junction, though, in a small number of modern humans (~3-5%) the presence of a 6th lumbar vertebrae has been noted (Lovejoy, 2007). The vertebrae consist of the vertebral body, which is the major load bearing component, and the vertebral arch consisting of the pedicle, transverse process, lamina, spinous process and superior and inferior articular processes. The pedicles and lamina form the lateral pillar and roof of the spinal canal protecting the spinal cord and proximal spinal nerves, and the combined

elements of the vertebral arch serve as attachments for muscles and ligaments, levers for muscular contraction to act against, and articulations with adjacent vertebrae (Drake et al., 2008).

The vertebrae articulate with one another through two joint types; the symphases between the vertebral bodies (intervertebral discs), and the synovial joints between the articular processes (zygapophyseal or facet joints). The intervertebral discs join adjacent vertebrae by means of a thin layer of hyaline cartilage (known as the endplate) and are composed of the nucleus pulposus (the gelatinous center providing hydrostatic properties to changes in pressure) and the annulus fibrosus (composed of the outer fibers of the lamellae which are differentially oriented in adjacent lamellae; Adams et al., 2010). The facet joints are where the two articular processes meet and are enclosed by a thin articular capsule. A number of ligaments also provide passive stability to the vertebrae including the anterior and posterior longitudinal ligaments surrounding the vertebral body, and the interspinous ligaments including the ligament flava and spinous ligaments (Drake et al., 2008).

The lumbar musculature, include the superficial Erector Spinae (ES; i.e. iliocostalis lumborum and longissimus thoracis) and both deep and superficial lumbar Multifidus (MF), both of which provide stability to the lumbar spine (Donisch & Basmajian, 1972; Bogduk & Twomey, 1987; Panjabi et al., 1989; Crisco & Panjabi, 1991; Cholewicki et al., 1997; Solomonow et al., 1998; Moseley et al., 2002; MacDonald et al., 2006; Rosatelli et al., 2008). These muscles originate at the sacrum, spinous processes and iliac crest and are covered by the thoracolumbar fascia (Drake et al., 2008) which has recently been highlighted to also provide a contributory role in spinal stability, static posture and movement (Willard et al., 2012). The muscles receive innervation from the posterior rami of the lumbar spinal nerves (Drake et al., 2008).

The complexity of the structures within the lumbar spine presents a number of potential mechanisms for pain originating from the area. Indeed many of the structures noted have been evidenced to be implicated in LBP.

1.4 Mechanisms of Pain within LBP

As highlighted, chronic LBP is multifactorial and a variety of symptoms/dysfunctions associated with it might cause pain due to stress they exert upon structures of the lower back known to elicit a pain response (Farfan & Gracovetsky, 1984). Pain is defined by the International Association of the Study of Pain (IASP) as “*an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.*” (IASP, 1994). Further, pain can be broken down into three components (Loeser, 1980); nociception, pain itself, and emotional suffering. Nociception refers to the process by which the nervous system senses noxious stimuli, pain itself is a central output relating to the perception of this sensation, and suffering is an emotional response to the pain output, or a false perception of a pain output. Such additional clarification of the definition implies that, though pain itself is a subjective experience, in general it has an organic source, or a potential organic source. However, despite there being a range of sources potentially responsible for pain perception (Salzberg, 2012), the possibility of pain characterised as emotional suffering arising without an organic source, or indeed absence of pain despite tissue damage, cannot be dismissed entirely.

First and foremost pain is regarded as a necessary response for survival of an organism. However, as DeLeo & Winkelstein (2002) distinguish, pathological pain processing should be considered differently from acute physiological pain processing. The Cartesian model of pain was described by Rene DesCartes in 1664 to present injury as a simple feedback system whereby the brain receives sensory input from the periphery (nociceptors) in response to noxious stimuli and processes it (DesCartes, 1664). This model is useful in describing acute physiological pain in normal populations, but pain, by definition, is a complex mechanism that may also be influenced by psychological mechanisms affecting

perception including prior experience, the context of the noxious stimuli, and also other physiological phenomena that may alter the nociceptive input (Price, 2000; DeLeo & Winkelstein, 2002; Apkarian et al. 2005). This has led to a biopsychosocial perspective being adopted to better explain the complex relationships between nociception, pain and suffering (Engel, 1980, Turk & Okifuji, 2002; Gatchel et al., 2007). Affective disorders can have significant impact upon pain response particularly when combined with actual tissue damage or other chronic pain states (Carragee et al. 2000). Evidence suggests that chronic pain states such as LBP are characterised by sensitisation of the central nervous system (CNS; DeLeo & Winkelstein, 2002; Steiger et al., 2012^a) and also the peripheral nervous system (PNS) (Hirsch et al. 1963; Edgar, 2007). Recent research examining structural differences in gray matter between age-matched chronic symptomatic and asymptomatic participants revealed chronic pain states are characterised by subcortical reorganisation (reduced brainstem and somatosensory cortex gray matter), suggested as a result of on-going nociception (Schmidt-Wilcke et al., 2006). Indeed neurons maintain a significant degree of plasticity allowing phenotypic changes which subsequently can alter the course of the pain response (Woolf & Salter, 2000).

In reviewing animal models, DeLeo & Winkelstein (2002) highlight that two specific local injury site mechanisms have been established to explain spinal pain etiology: 1) mechanical deformation of nerve roots and 2) biological or biochemical activity of herniated disc tissue. They consider the injury biomechanics by which the two specific pain mechanisms might be initiated highlighting that injury severity is closely related to both PNS sensitisation by degree of nerve root damage, and CNS sensitisation by up-regulation of inflammatory modulators and cellular responses dependent upon degree of tissue deformation. They note that nerve root swelling correlates well with initial injury magnitude, but it does not explain temporal patterns observed in behavioural sensitivity which is possibly due to differential modulation of downstream inflammatory processes in the PNS and CNS. Thus, beyond a certain degree of injury severity, peripheral cellular responses may not differ, whereas inflammatory processes may, potentially explaining the

range of individual responses in LBP and the marked differences in presence or absence of lumbar spine abnormalities in diagnostic tests and associated perception of pain.

Tissue damage can cause pain (Siddall & Cousins, 1997; Woolf et al. 1998; McGill, 2007), however, numerous studies have highlighted abnormalities in asymptomatic participants using a variety of diagnostic methods including myelograms (Hitselberger & Witten, 1968), magnetic resonance imaging (MRI; Boden et al. 1990; Jensen et al. 1994; Weishaupt et al. 1998; Borenstein et al. 2001; Barento et al. 2009; Maurer et al. 2011), radiography (Iwamoto et al. 2005) and discography (Holt, 1968; Walsh et al. 1990). This has brought into question the likelihood of such findings in symptomatic patients holding any clinical relevance to the pain they are experiencing. However, it should be considered, as highlighted (DeLeo & Winkelstein, 2002), that the subsequent pain response after initiation of injury is partially dependent upon its severity. In the case of abnormalities in asymptomatic participants it may be that a lower threshold may not have been achieved to initiate perception of a pain response. Though, in chronic LBP, pain sometimes manifests out of proportion to identified tissue damage (Garland, 2012). Another consideration should be the fallacy of logical inference from absence of evidence to indicate evidence of absence in symptomatic participants. An imaging study may show no obvious abnormalities in a symptomatic participant, however, it may have missed something, been misinterpreted, or whatever caused, or is still causing pain is just not visible on the particular test used. Both McGill & Yingling (1999) and Zhao et al (2005) highlight that diagnostic tests can be influenced by a variety of factors resulting in a false negative result. It may be possible that, in symptomatic participants without abnormalities, or with minor tissue damage, previous injury may have occurred before imaging which was not documented or recalled and which may have caused either PNS or CNS sensitisation, and thus, chronic pain. These cases should not be used to infer however that all chronic pain is necessarily independent of any form of tissue damage in the wider population.

Specific abnormalities, and the likelihood the structures they refer to are indeed potentially pain causing, should also be considered. Weishaupt et al. (1998) identified that disc bulging or protrusion, and degeneration, were more common than other more severe abnormalities such as herniation, end plate abnormalities, nerve root compression and facet joint osteoarthritis in asymptomatic individuals. Holt (1968), in a paper highlighting the issue of false positive results from discography in asymptomatic participants, dismissed the finding in his data that although discography showed cases of degeneration in 37% of asymptomatic participants, discography produced LBP or sciatic symptoms in all cases of herniation. Walsh et al. (1990) replicated this study also including a symptomatic group as comparison. They found significantly greater abnormal discographs and pain responses in the symptomatic group; however they did not report any distinction between degeneration and herniation. The intervertebral disc itself has a nociceptive nerve supply (Hirsch et al., 1963) that penetrates deep into the nucleus pulposus. This appears to be hyperalgesic in participants with degenerated discs compared to that of normal discs (Edgar, 2007). Perhaps, considering these findings, the degree of disc deformation imparts some influence on pain (Holt, 1968). In asymptomatic participants Schmorl's nodes (Intervertebral disc protrusion through the endplate and into the vertebral body, initiating an inflammatory response and Modic changes i.e. degeneration of vertebral bone marrow) are far less common than all disc abnormalities with the exception of herniation (Jensen et al. 1994). Herniation (Albert & Manniche, 2007), end plate fracture and degeneration (De Roos et al. 1987; Toyone et al. 1994) have been shown to be risk factors for modic changes, instigating an inflammatory and thus potentially pain causing response (McCarron et al. 1987; Saal et al. 1990). Indeed Kjaer et al. (2005) support this highlighting the strongest positive associations between chronic LBP and disc abnormalities to be for modic changes, whilst only moderate associations were found between all other disc abnormalities. It would seem that, as DeLeo & Winkelstein (2002) suggest, the severity of tissue deformation/degeneration and injury, particularly in the disc, has an impact upon whether or not pain is present as a symptom and potentially the degree of pain experienced. Recent studies also support the contention that more severe

degrees of degeneration, structural abnormality, or a combination of the both are more consistently apparent in participants with chronic LBP than in those who are asymptomatic in a dose dependent manner (Cheung et al., 2009; de Schepper et al., 2010). Even if not all abnormalities can be ascribed as the source of chronic LBP, any degenerative changes also heighten the risk for more severe disc degeneration or injury and thus potentially pain and suffering (Adams et al., 2010).

Direct nerve or disc injuries are not the only mechanisms which can initiate a pain response. Bogduk (1997) suggests that for a structure to be deemed a potential cause of pain it must meet four factors: have a nerve supply; be identified to cause pain similar to that seen in clinical scenarios in normal volunteers; be susceptible to painful disease or injury; and be demonstrated as a source of pain using diagnostic techniques. Facet joint pain is prevalent in chronic LBP and based on these criteria is well established to be a potential source of pain (Manchikanti et al. 1999; Manchikanti et al. 2004). In addition the ligamentous tissue of the lumbar region may be a potential source for pain as abnormalities are more common in symptomatic participants (Fujiwara et al. 2000) and they are shown to contain a rich free nerve supply suggesting injury could contribute to pain (Hirsch. 1963; Kiter et al. 2010). However, the lumbar musculature may not necessarily be a direct source of pain due to the persistence of muscle abnormalities after symptoms have resolved in first case acute LBP (Hides et al. 1996). Though, due to its active role in providing stability, the musculature may indirectly be responsible for instigating injury and pain causing mechanisms in the other structures (Donisch & Basmajian, 1972; Bogduk & Twomey, 1987; Panjabi et al., 1989; Crisco & Panjabi, 1991; Cholewicki et al., 1997; Solomonow et al., 1998; Moseley et al., 2002; MacDonald et al., 2006; Rosatelli et al., 2008).

A hypothetical lower threshold of nerve root deformation, and it seems potentially disc deformation also, is proposed for initiation of pain behaviour (DeLeo & Winkelstein, 2002), however, in light of muscular models of spinal stability (Donisch & Basmajian, 1972;

Bogduk & Twomey, 1987; Panjabi et al., 1989; Crisco & Panjabi, 1991; Cholewicki et al., 1997; Solomonow et al., 1998; Moseley et al., 2002; MacDonald et al., 2006; Rosatelli et al., 2008), it might be hypothesised that in the normally functioning lumbar spine, nerve root compression or other injury should not occur unless something has caused instability and altered joint biomechanics e.g. abnormal muscle function and/or injury. DeLeo and Winkelstein (2002) hypothesise that injury initiates a cascade of events, which may not be linear, but that ultimately contribute to a pain response and may alter over time due to the interrelated influence of such events. Indeed the non-linearity of such events (Brinkmann, 1985; Butler et al., 1990; Kirkaldy-Willis and Burton, 1992) may be due to the non-linear effect of distal primary injury upon proximal secondary injury/pain mechanisms (Whiting & Zernicke, 1998). Panjabi's (2006) hypothesis of the non-linear sequential events after sub-failure injury concisely describes how these events might be highly variable.

Whiting & Zernicke (1998) in discussing injury and pain highlight the important point that mechanisms should not be confused with the related albeit different concept of predisposing or contributory factors (i.e. factors that increase the chance of a mechanisms initiation). Most LBP is likely the result of some form of injury initiating a pain mechanism, the specific injury mechanisms being an intermediary factor (though it is acknowledged that pain may arise independent of injury and an organic nociceptive source). What should be considered, as mentioned, are the factors contributing to the initiation of these subsequent mechanisms. It seems the majority of LBP appears to have an organic pain causing origin. Although, LBP may be psychosomatic (perhaps better defined in these cases as 'suffering' considering Loeser's (1980) aspects of pain) in some instances, and certainly psychological factors might affect the degree of pain and suffering experienced. However, most epidemiological evidence for this is affected by confounding factors (Punnett & Wegnam, 2004) and considered to be primarily circumstantial (Smeets et al. 2006). DeLeo and Winkelstein (2002) suggest that understanding of spinal and supraspinal mechanisms, and mediators of CNS sensitisation could help direct treatment. It should be acknowledged though that the best approach for any condition is prevention,

and that in the context of an existing condition, treating the causative mechanism or predisposing factor might also result in favourable outcomes. In the case of addressing such downstream consequences shown to be initially caused by injury, we should ask whether or not it is more prudent to instead address what caused injury in the first place in order to allow healing to occur.

1.5 A Multifactorial Perspective of Low Back Pain

As noted a number of potentially pain causing mechanisms exist in LBP, though, it is not always possible to directly attribute pain to a specific source through diagnostic means. Because of this it is not always possible to monitor improvement in an underlying cause, pain mechanism or diagnosis. As such, changes in function are often used to indicate improvement.

Function often focuses on the musculoskeletal system, due to its proposed implication in LBP, and thus has considered strength, endurance and ROM (Plowman, 2001). Adding to this Lee and Vleeming (1998, 2007) have suggested the conceptualisation of LBP within an integrated multifactorial framework of function in relation to the spine and pelvic girdle (Figure 1). Function in the context of LBP therefore comprises several factors. The array of dysfunctions in LBP could be considered as interrelated deficiencies in function under the areas of; form closure (structure), force closure (force produced by myofascial action), motor control (neural patterning and control), and emotions (psychological and psychosocial factors). Lee & Vleeming (1998; 2007) posit that LBP and the function of the lumbar spine are linked, that its various commonly associated symptoms are potentially interrelated dysfunctions together resulting in the condition, and that improvement in function should be considered in a range of areas. Indeed this conceptualisation is in agreement with the range of dysfunctions and pain causing mechanisms discussed in the previous section and the non-linearity of their responses. Further, Lee & Vleeming (1998, 2007) suggest that conceptualising LBP within this framework is valuable for hypothesis generation regarding both understanding etiological factors influence upon its

multifactorial nature, in addition to suggesting that the efficacy of treatments should be evaluated by considering a range of functional outcomes.

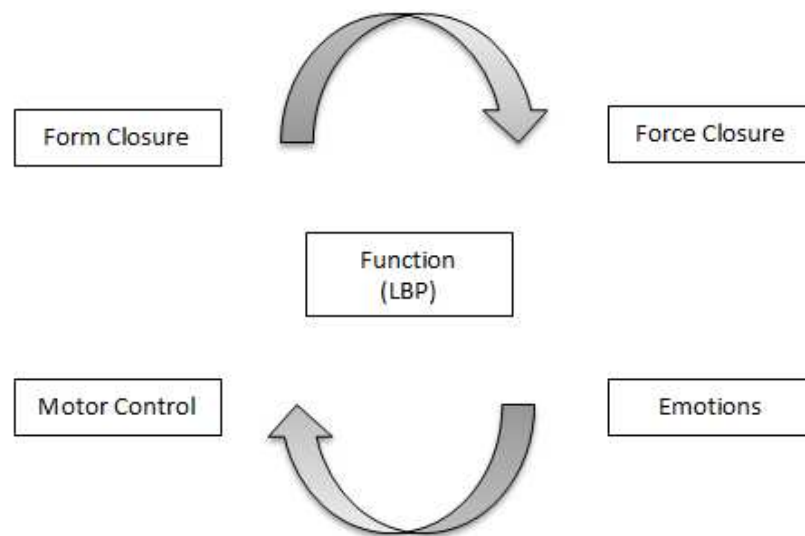


Figure 1. Integrated multifactorial framework of function (Lee & Vleeming, 1998)

The population specifically considered in this thesis are those suffering from LBP that are categorised as non-specific (NICE, 2009). It has been suggested that there is potential benefit of sub-categorisation of LBP based upon symptom presentation (Lebouef-Yde et al., 1997; Hepple & Robertson, 2006). In light of the multifactorial nature of LBP sub-categorisation might offer considerable value. Many clinicians consider it a valid concept that should direct treatment when considering non-specific chronic LBP; however, there is little validating evidence to support it³ (Kent & Keating, 2004). Indeed research, although suggesting that imaging abnormalities are more common in symptomatic as opposed to asymptomatic participants, has found that they rarely bear any differential relationship to individual clinical outcomes (i.e. pain and perceived disability) thus offering little prognostic value for determining treatment (McNee et al., 2011; Shambrook et al., 2011; Endean et al., 2011). Though it may not yet offer value in directing treatment choice within a clinical setting, conceptualisation using a multifactorial framework instead represents a useful tool in directing research into etiology and for considering a range of outcomes regarding

³ Despite the problems with sub-categorisation and determination of pain causing symptoms in non-specific LBP it should be noted that in rehabilitation some clinical symptoms may be contraindicative to particular treatment options, such as the one utilised in this study (Isolated lumbar extension resistance training). This was an important consideration during determination of exclusion criteria for the studies conducted towards this thesis and is detailed further within the methods chapter.

function as a result of intervention. Thus, this thesis has used this conceptualisation in its examination of the role that a specific aspect of function plays both etiologically and as a treatment target; the lumbar extensor musculature.

2. LITERATURE REVIEW

2.1 Introduction and Overview

Considering that LBP appears to be most predominantly caused by injury or degeneration to one or many of the structures of the lumbar spine, its stability and ability to resist external loading seems a promising predisposing factor to examine as playing a role in low back injury risk and LBP. Indeed as noted, the extensor musculature have been demonstrated to be predominantly involved in maintaining such stability (Donisch & Basmajian, 1972; Bogduk & Twomey, 1987; Panjabi et al., 1989; Crisco & Panjabi, 1991; Cholewicki et al., 1997; Solomonow et al., 1998; Moseley et al., 2002; MacDonald et al., 2006; Rosatelli et al., 2008) and their condition could impact upon low back injury risk and pain. Further, how to target and condition the musculature in the most efficacious manner may also be justified in examination. Therefore the following sections of this literature review chapter examine the following;

1. The etiological role of lumbar extensor muscle deconditioning in LBP.
2. Which exercise based approaches might be most efficacious in conditioning the lumbar extensor musculature.
3. What impact such exercise based approaches addressing the lumbar extensor musculature have upon pain and disability in symptomatic persons.

These sections are presented as independent systematic reviews whereby an initial independent introduction is provided for each section followed by methods, including search strategy and inclusion criteria, results of the literature review, and a discussion and conclusion offered. The first three sections have been previously published in peer reviewed journals with the present author as first author. These sections therefore are presented in the same format as their published versions⁴ and thus are written in a manner independent from the remainder of the thesis.⁵ However, at the end of the

⁴ With the exception of the heading and referencing formats being amended to the required format for this thesis.

⁵ It should be noted that, due to the order in which content of this thesis was accepted for publication, some of these reviews refer to the first of the empirical studies performed towards this thesis (i.e. the first empirical piece was accepted for

literature review chapter a section synthesising the evidence and conclusions from these independent reviews is offered providing an overarching rationale for the thesis. From this the specific areas of empirical research are presented and literature reviewed offering justification for their investigation within the context of the overarching thesis rationale, in addition to the research questions and hypotheses this thesis aims to address.

2.2 A Reappraisal of the Deconditioning Hypothesis in Low Back Pain: Review of Evidence from a Triumvirate of Research Methods on Specific Lumbar Extensor Deconditioning⁶

2.2.1 Overview⁷

‘Disuse’ and ‘Deconditioning’ in relation to low back pain (LBP) are terms often used interchangeably. Discussions of ‘disuse’ refer to general physical inactivity, which evidence suggests does not differ between symptomatic and asymptomatic persons. ‘Deconditioning’ refers to a decrease in function, commonly both cardiovascular/aerobic fitness and muscular strength/endurance again noting little difference. However, examination of decreased function relating specifically to lumbar extensor musculature deconditioning has yet to be examined corroborating all possible methods. Thus, this review attempts to reappraise the deconditioning hypothesis in LBP specifically considering lumbar extensor deconditioning. A literature review was conducted examining both cross sectional and prospective data on specific lumbar extensor deconditioning and LBP. A narrative approach and ‘snowballing’ style literature search was used involving initial use of PubMed and Google Scholar databases searching up to December 2012. Included were studies utilising the following three research methods allowing specific induction of the role of such deconditioning; 1) strength/endurance testing of the isolated lumbar extensor musculature, 2) imaging and histochemical examination of the lumbar extensor musculature, and 3) fatigue testing of the lumbar extensor musculature using electromyography. Despite issues interpreting individual studies due to methods, the majority of evidence suggests LBP is associated with decreased strength/endurance, atrophy, and excessive fatigability of the lumbar extensors. Prospective studies also suggest lumbar extensor deconditioning may be a common risk factor predicting acute low back injury and LBP. The hypothesis of specific lumbar extensor deconditioning as being a causal factor in LBP is presently well supported. It is by no means the only causative

⁶ Note that this section has been previously published as an independent article by the author; Steele et al., 2013. A Reappraisal of the Deconditioning Hypothesis in Low Back Pain: Review of Evidence from a Triumvirate of Research Methods on Specific Lumbar Extensor Deconditioning. *Current Medical Research and Opinion*. 30(5), pp 865-911.

⁷ Overview = Abstract amended from the published version.

factor and further research should more rigorously test this hypothesis addressing the methodological issues highlighted regarding previous studies. However, its role suggests specific exercise may be a worthwhile preventative and rehabilitative approach.

2.2.2 Introduction

2.2.2.1 Defining the Disuse/Deconditioning Hypothesis - ‘Disuse’ OR ‘Deconditioning’?

The ‘Disuse Syndrome’ was originally described by Bortz II (1984) and more recently has been reviewed by Verbunt et al. (2003; 2010). The rationale behind Disuse Syndrome is that pain causes low levels of physical activity (i.e. avoidance behaviour or guarded movement; Main and Watson, 1996) which contribute to deconditioning and chronicity in low back pain (LBP), and cause the further interrelated physical and psychological changes shown in figure 2. In essence, it proposes that injury and pain precede deconditioning and potentially many of LBP’s symptoms, leading to a ‘vicious cycle.’

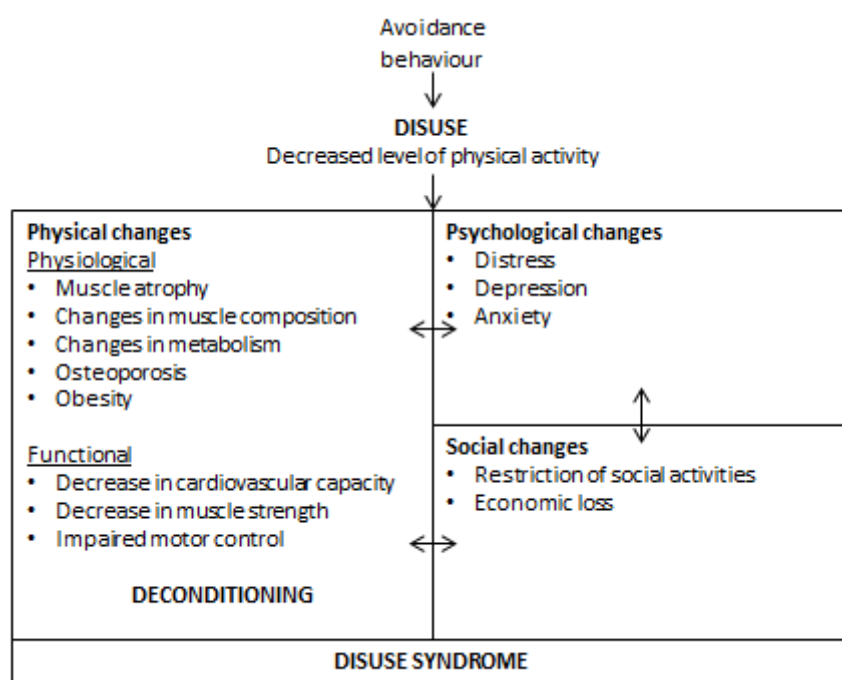


Figure 2. Disuse Syndrome Model, from Verbunt et al. (2003)

Verbunt et al. (2003) however, have suggested that the hypothesis that ‘disuse’ (i.e. defined as a *decrease in physical activity levels*) is a cause of LBP may be incorrect. They highlight that activity levels are in fact similar between symptomatic and asymptomatic

participants, suggesting that lack of physical activity due to the presence of pain or injury may not contribute to the presence of deconditioning or LBP (Verbunt et al., 2001). Indeed a more recent study has also highlighted that physical activity levels appear to not change as a result of LBP, even as it develops from acute into chronic LBP (Bousema et al., 2007). This suggests that symptomatic chronic LBP participants may not suffer from development of disuse after the initial incidence of LBP. It seems possible, therefore, that the direction of temporal relationships in the 'Disuse Syndrome' model may be unfitting in its usual presentation (figure 2). This is not to suggest that deconditioning as a result of existing pain and its related behaviours is not a possibility, indeed the presence of injury has been shown to affect muscular function and could therefore instigate deconditioning itself, or at the least further enhance its development (Mannion, 1999; Panjabi, 2006).

Instead, 'deconditioning' (i.e. defined as a *decrease in function*) may be first implicated as a potential cause of low back injury and pain, as opposed to LBP leading to 'disuse' and then to 'deconditioning'. Indeed Verbunt et al. (2003) attempt to distinguish between 'disuse' and 'deconditioning'; however in both their definitions they inevitably invoke general physical inactivity ('disuse') as being responsible for 'deconditioning' and that this inactivity is the result of pain. Here we instead differentiate between 'disuse' and 'deconditioning' and pose that the disuse syndrome model does not consider what first causes or increases the probability of injury or pain occurring (which *may* lead to further 'disuse') in the first place.

In addition, the disuse model appears to imply that freak injury may account for the majority of LBP (Mannion, 1999), yet due to the widespread prevalence of LBP it seems unlikely that freak injury could account for the majority of cases. Bigos et al. (1986) demonstrate exactly why this is a concern, reporting that accidents such as slips or falls, despite resulting in higher cost injuries, are very uncommon with regard to cause of injury; lifting or materials handling, however, was most commonly considered a cause. Dysfunction due to deconditioning could potentially affect such actions leading to fatigue

and altered joint biomechanics, subsequently causing injuries and instigating mechanisms by which pain results. Then, at this point, the cycle by which a reduction in activity levels further promote chronicity, and the changes associated with it, may begin to have an influence.

The model may therefore require an addition that considers the initial injury in the first place (figure 3). A high percentage of low back injury and acute LBP develops into chronic LBP (Papageorgiou et al., 1996; Croft et al., 1998) and so it seems logical that something must affect the risk of low back injury in the first instance. Indeed Adams and colleagues (2010) have recently commented on the pertinent fact that all *'chronic back pain always starts as acute back pain.'* Thus, logically, something must first be responsible for the initiation of acute pain. The remainder of the existing model is likely correct in describing the process of developing chronicity after initial injury has occurred. What specific factor is most important in determining whether that initial injury and acute pain occurs in order for it to become chronic, however, is the more interesting question.

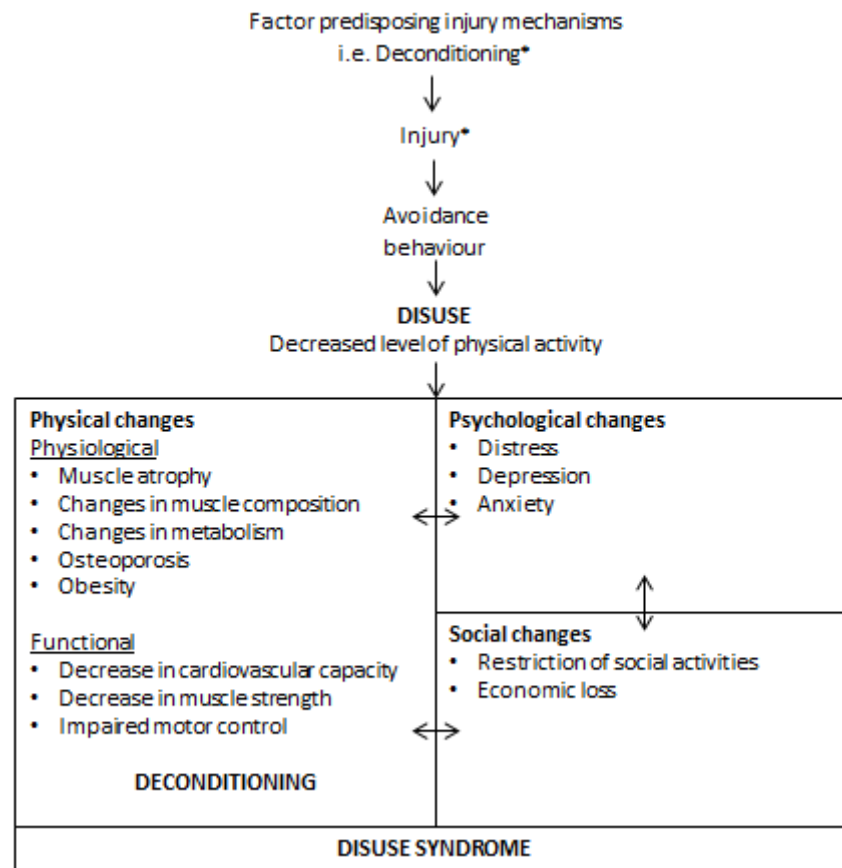


Figure 3. Deconditioning Syndrome Model – Adapted* Disuse Syndrome Model, adapted from Verbunt et al. (2003)

Many prior reviews on the topic of deconditioning in LBP have utilised a broad focus encompassing decrease in function of both cardiovascular/aerobic fitness as well as muscular strength/endurance (Verbunt et al., 2003; 2010; Wittink et al., 2000; Smeets et al., 2006). These reviews suggest that deconditioning of these kinds is not apparent in those with chronic LBP. However, the studies considered have utilised varied methods of examining this association, many of which are not entirely specific and these are explained throughout this article. The lack of consideration of the specific study methodologies used by previous authors has perhaps contributed to the vague distinctions between ‘disuse’ and ‘deconditioning’ as well as the shift in focus from physical risk factors towards a more cognitive based appraisal of LBP and the effects of rehabilitation (Steiger et al., 2012^a). A more specific analysis of the literature, focusing upon specifically located deconditioning and in consideration of the methodological

limitations of prior techniques, might therefore be found to yield contrasting conclusions regarding the presence of deconditioning in LBP and the models relationships.

2.2.2.2 Specific Disuse/Deconditioning of the Lumbar Extensors

The important role of the lumbar extensor musculature, the Erector Spinae (ES; i.e. iliocostalis lumborum and longissimus thoracis) and both deep and superficial lumbar Multifidus (MF), in providing stability to the lumbar spine, has been alluded to in numerous studies (Donisch & Basmajian, 1972; Bogduk & Twomey, 1987; Panjabi et al., 1989; Crisco & Panjabi, 1991; Cholewicki et al., 1997; Solomonow et al., 1998; Moseley et al., 2002; MacDonald et al., 2006; Rosatelli et al., 2008). Prior reviews of the literature have indicated that, although it is difficult to distinguish which muscles provide the greatest relative contribution to spinal stability, their importance in co-operatively contributing to lumbar spinal stability is clear (MacDonald et al., 2006; Cholewicki & Van Vliet, 2002). The relative contribution of individual muscles will vary depending on the specific task being performed (Ladin et al., 1989); however, MacDonald et al. (2006) explain that all lumbar extensors can contribute towards stability of the intervertebral segments through compression of the vertebral unit and increased joint stiffness. Clearly the lumbar extensor musculature playing such an important role in providing stability to the lumbar spine suggests that deconditioning and dysfunction in these muscles could lead to changes in stability and biomechanics. This change in biomechanics may result in increasing passive tissue stresses and potentially impart an injury or pain response in structures of the lumbar spine (Farfan & Gracovetsky, 1984).

2.2.2.3 Aim and Approach of this Review

The focus upon 'disuse' and 'deconditioning' in a general sense has led to much incongruity in drawing specific conclusions regarding LBP. In light of the potential role of the lumbar extensors in controlling stability and, in dysfunction, altering biomechanics which might lead to injury and pain causing mechanisms/symptoms, there is certainly potential for specifically located deconditioning to relate to LBP. The aim of this review

therefore is to test this hypothesis by examining the evidence reporting the nature of the relationship between specific deconditioning of the lumbar extensors and LBP from a triumvirate of research methods including:

- Strength/endurance testing of lumbar extensor musculature,
- Imaging and histochemical examination of the lumbar extensor musculature
- Fatigue testing of the lumbar extensor musculature using electromyography

Three sections will follow, each covering the three research methods highlighted as they have been used in cross sectional examination of symptomatic chronic LBP participants compared with asymptomatic healthy participants. A fourth section shall examine prospective studies that have sought to examine the effect of deconditioning using these methods upon development of LBP in asymptomatic participants. Throughout, any methodological concerns and considerations with studies shall be highlighted initially and noted whilst discussing such studies. In light of those methodological issues discussed in each section it will also be noted which types of studies were excluded from consideration (however, those excluded are still summarised within the full summary tables in the appendices⁸). Given the broad scope of this review a narrative approach utilising a 'snowballing' style literature search (Greenhalgh & Peacock, 2005) was used initially involving PubMed and Google Scholar databases searching up to December 2012 utilising search terms including combinations and synonyms of 'low back pain' low back injury' 'lumbar' 'back' 'spine' 'extensors' 'lumbar extension' trunk extension' 'erector spinae' 'multifidus' 'iliocostalis lumborum' 'longissimus thoracis' 'strength' 'endurance' 'atrophy' 'cross sectional area' 'fat infiltration' 'muscle density' 'histochemistry' 'fibre type' 'electromyography' 'fatigability' etc. In addition previous reviews and any located articles reference lists were searched. This was selected as the best way to locate, examine and synthesise the maximum amount of information in the various sections covered, thus initial inclusion criteria were based upon applicability to this particular area of discussion,

⁸ See appendix 7.5

and whether studies had utilised the above noted research methods, before applying specific exclusion criteria (noted in each section of this review).

2.2.3 Strength and Endurance of the Lumbar Extensor Musculature in LBP

2.2.3.1 Considerations for Studies of Strength and Endurance of the Lumbar Extensor Musculature

An initial consideration when looking at studies of muscular performance should be that of the false duality between definitions of muscular strength and endurance expressed by many authors especially within the field of exercise and LBP (McGill, 2007; Norris, 2008). Muscular endurance can be defined as being *absolute* (i.e. the number of repetitions/time performed at a given resistance), or *relative* (i.e. the number of repetitions/time performed at a given percentage of a 1 repetition maximum [1RM] or other maximum strength measurement; Stone & Coulter, 1994; Carpinelli et al., 2004; Fisher et al., 2011). For example, a pre training 1RM of 100kg might produce 10 repetitions at an absolute value of 70kg, which is also the relative value of 70%1RM. However, after a training intervention where the 1RM has improved to 120kg, a participant will almost certainly be capable of greater than 10 repetitions at the absolute value of 70kg, but likely still only produce a maximum of 10 repetitions at the relative value of 70% 1RM (now 84kg). This example shows an increase in maximal strength (1RM) leading to an increase in absolute muscular endurance (i.e. an increase in number of repetitions at the fixed submaximal weight). Research supports this concept (Hickson et al., 1994). However, research does not support the idea that the same is true of relative loads, but rather that similar maximal repetitions are possible (Hickson et al., 1994; Mazzetti et al., 2000). In practice, with relevance to the deconditioning hypothesis and for the LBP participant, low strength would translate to low absolute endurance, high strength to high absolute endurance and vice versa. Therefore, presuming average external loads typically experienced (i.e. in working conditions etc.) might remain constant, an increase in strength would theoretically mean an increase in endurance at those absolute loads experienced. Thus it would seem logically erroneous to attempt to draw a distinction between the two and to claim that one

is more important than the other with regards to LBP (McGill, 2007). Indeed Mannion (1999) has commented that the hypothesis of fatigability as being associated with LBP is essentially analogous to the hypothesis of insufficient strength (both being manifestations of lumbar extensor deconditioning relevant to the deconditioning hypothesis).

Numerous studies have sought to identify the relationship between functional measures of strength and endurance of the lumbar extensor muscles. However, it should be highlighted that the validity of a number of methods of tests for strength and/or endurance of the lumbar spine are questionable due to methodological difficulties; a primary concern being whether sufficient pelvic restraints have been utilised. Essentially, tests have either been performed to examine *trunk* extension (TEX), OR, isolated *lumbar* extension (ILEX; testing utilising pelvic stabilisation through use of a semi seated position with rear pelvic restraint and a belt across the thighs). In considering lumbar extensor deconditioning TEX studies require careful reflection along with corroboration of more valid test measures i.e. ILEX. If the pelvis is not stabilised during testing of extension then it is impossible to determine the actual source of measured extension torque during tests of strength and may involve the hip extensors (Kankaanpaa et al., 1998^a; Kankaanpaa et al., 1998^b; San Juan et al., 2005; Da Silva et al., 2009; Lariviere et al., 2010^a; Clark et al., 2002; 2003) contributing to overstate torque measures (Smidt et al., 1983; Petersen et al., 1987; Graves et al., 1992^a), due to the longer moment arms over which the gluteus and hamstrings exert force, and their relatively larger cross-sectional areas (Farfan, 1975). At most only 3° of pelvic rotation (Inanami, 1991), likely a result of soft tissue compliance, occurs during ILEX testing of this kind. Lack of pelvic restraint perhaps partly explains the inconsistent reproducibility of TEX endurance tests (Alaranta et al., 1994; Moffroid et al., 1994; Mayer et al., 1995; McGill et al., 1999; Latimer et al., 1999) as compared with the consistency of ILEX strength and endurance testing (Graves et al., 1990^a; Robinson et al., 1992^a; Udermann et al., 2003; Hager et al., 2006). Indeed, despite the aforementioned relationship between strength and absolute endurance there is poor relationship between

tests of ILEX strength and TEX endurance (Perez et al., 2007). This highlights that the tests may utilise different musculature.

Although tests of ILEX are more valid representations of lumbar extensor function due to TEX being a compound movement requiring additional rotation of the pelvis through the hip extensor musculature (Kankaanpaa et al., 1998^a; Kankaanpaa et al., 1998^b; Clark et al., 2002; 2003; Smidt et al., 1983; Petersen et al., 1987; Graves et al., 1992^a; Farfan, 1975; Inanami, 1991; Fulton, 1993; Pollock et al., 1993; Smith et al., 2008), a large number of studies have made use of tests measuring TEX. Smidt et al. (1983) also explain that consideration of both tests of TEX and ILEX are indeed valuable when interpreted together as they allow both a deductive, and further an inductive, approach to identify the so called 'weak link' within the kinetic chain and thus we initially considered both studies in this review. Beimborn & Morrissey (1988) reviewed early literature on trunk muscle performance in LBP suggesting a consistent association with reduced TEX strength in symptomatic participants, as well as further studies. These TEX studies have been summarised in the appendices⁹ provided and appear to show inconsistent associations; some results supporting a link between TEX strength/endurance and LBP some which do not.

The inconsistency of both TEX tests of strength and endurance should not be surprising as hip extensor deconditioning appears to not be associated with chronic LBP (Kamaz et al., 2007) and as explained, without appropriate restraint of the pelvis the musculature of the hip extensors will serve to confound results. However, despite hip extensor deconditioning having apparently little association with LBP it seems some other aspect of TEX, perhaps ILEX, may be associated with it. As TEX is composed of both hip and lumbar extension it therefore seems logical that tests should attempt to remove the involvement of the hip extensors to examine ILEX. Thus, of key importance and inclusion to this review are studies that have used appropriate methods of testing ILEX. As shall

⁹ See appendix 7.5.1

also be noted, previous surgery may have implications for the results of studies examining the deconditioning hypothesis (Weber et al., 1997; Rantanen et al., 1993; Sihvonen et al., 1993; Motosuneya et al., 2006) and ideally participants with prior surgery should be excluded. However, only one study utilising ILEX has controlled for this factor yet may suffer from its own shortcoming of small sample size. Thus in the following section all ILEX studies have been examined with this limitation noted.

2.2.3.2 Isolated Lumbar Extension Studies

The validity of the extension test used is of great importance in examining the association between strength, endurance and LBP, therefore studies that have considered this are potentially more useful in answering the question of whether specific lumbar extension deconditioning is associated with LBP. Unfortunately in comparison with studies of TEX, studies of ILEX are relatively scarce. However, studies utilising testing that appropriately restrains the pelvis consistently report significantly reduced ILEX strength in symptomatic chronic LBP participants compared with asymptomatic controls (Cassisi et al., 1993; Holmes et al., 1996; Robinson et al., 1992^b). Other studies (Nelson et al., 1995; Mooney et al., 1995; Mooney et al., 1997; Boyce et al., 2008) have further reported reduced strength results from symptomatic participants compared to normal values obtained from healthy asymptomatic controls in other research (Graves et al., 1990^a).

There is, however, only one study by Lariviere et al. (2010^a) of ILEX using valid restraints that does not support the link between specific deconditioning and chronic LBP. Lariviere et al. (2010^a) reported no difference between asymptomatic and symptomatic chronic LBP participants in strength reported as maximum torque, or endurance reported as repetitions performed at a load equal to 60% maximum voluntary contraction (MVC). They commented however in discussion that the small sample size (n=18) used may have meant a lack of the typical multifactorial heterogeneity in their non-specific chronic LBP group, potentially impacting the generalisation to chronic LBP of this aspect of their research. It may also have resulted in a type II statistical error (i.e. failure to reject the null

hypothesis). Other larger studies that have supported the link between ILEX weakness and chronic LBP have used in some instances upwards of 100 symptomatic participants and demonstrate reduced strength compared to healthy norms (Nelson et al., 1995; Mooney et al., 1995). In addition and in particular, the study by Nelson et al. (1995) of 895 chronic LBP participants suggested that a range of diagnoses existed in their sample (Patients' diagnoses included non-specific chronic LBP, degenerative disc/arthritis disease, lumbar disc syndrome or spondylolisthesis/spondylolysis) and thus was likely quite representative of the typical heterogeneity of chronic LBP populations. Age, stature and body mass were also similar between groups in the study by Lariviere et al. (2010^a), however this was also reportedly the case for a number of other studies supporting the association (Cassisi et al., 1993; Holmes et al., 1996; Robinson et al., 1992^b) and so is unlikely to explain the difference in results.

One limitation of studies supporting the link between reduced ILEX strength and LBP is that these studies either did not report whether they excluded (Holmes et al., 1996; Nelson et al., 1995; Mooney et al., 1995; Mooney et al., 1997; Boyce et al., 2008), or chose not to exclude (Cassisi et al., 1993; Robinson et al., 1992^b), participants who had undergone previous surgery. Lariviere et al. (2010^a) did exclude those having undergone previous lumbar surgery and thus this may explain the different results found by these investigators. As has been noted, previous surgery can have potentially deleterious consequences to the lumbar extensor musculature anatomy (Weber et al., 1997; Rantanen et al., 1993; Sihvonon et al., 1993; Motosuneya et al., 2006) and so might be thought to interfere with ILEX strength in symptomatic participants. Although a number of TEX studies have excluded those with previous surgery, with some supporting and some refuting¹⁰ an association between deconditioning and LBP, we must consider the inherent limitations of this approach already highlighted when specifically concerned with the lumbar extensors. There is certainly potential for further research to clarify whether differences in ILEX strength do indeed exist independent of previous lumbar surgery.

¹⁰ See appendix 7.5.1

A final concern is the lack of statistical comparison with healthy controls groups in some studies (Nelson et al., 1995; Mooney et al., 1995; Mooney et al., 1997; Boyce et al., 2008). Though these results are consistent with those that have conducted statistical comparisons (Cassisi et al., 1993; Holmes et al., 1996; Robinson et al., 1992^b) this is a weakness and again something to be ensured in future research.

It is noted that only one study reported upon tests of isolated lumbar extension endurance (Lariviere et al., 2010^a). However due to the inherent relationship between strength and endurance it seems logical that the reported reduced lumbar extension strength in chronic LBP would be indicative of a reduced endurance also. The limitations discussed above also apply to this aspect of the study however, and there is further scope for research specifically examining this.

2.2.3.3 Summary of Strength and Endurance Studies of the Lumbar Extensor Musculature

Of the studies examined, those employing sufficient pelvic restraints as their means of assessing lumbar extension have consistently reported results that lend support to the association of specific lumbar extensor deconditioning with chronic LBP (Cassisi et al., 1993; Holmes et al., 1996; Robinson et al., 1992^b; Nelson et al., 1995; Mooney et al., 1995; Mooney et al., 1997; Boyce et al., 2008) with only one exception (Lariviere et al., 2010^a). It seems clear that when valid testing of ILEX is used, most evidence suggests a link between specific lumbar extension deconditioning and chronic LBP. However, it is unclear from purely this area of research whether this may in fact be due to the presence of previous surgery. Studies controlling for this factor utilising a larger sample size should be conducted to further test this. Table 1 summarises the findings of these studies.

Table 1. Summary of studies testing strength and endurance of the lumbar extensor musculature in LBP

Reference	Participants	Testing	Results	Comments
Lariviere et al. (2010 ^a)	Healthy controls without LBP lasting 1 wk in previous year, $n = 18$ CLBP patients, $n = 18$	Isolated lumbar extension MVC and number of repetition to failure at 60%MVC using customised dynamometer	No significant difference between groups	Those with previous lumbar surgery were excluded Age, body mass, height, BMI, body % and physical activity levels were similar between groups
Cassisi et al. (1993)	Healthy controls without history of LBP, $n = 12$ CLBP patients, $n = 21$	Isolated lumbar extension MVC using MEDX	Lumbar extension significantly weaker in CLBP ($p = 0.01$)	13 CLBP patients had undergone previous surgery though no effect upon lumbar extension strength was observed Age and height were similar between groups though body mass was greater in CLBP group
Holmes et al. (1996)	Healthy geriatric female controls, $n = 20$ CLBP geriatric female patients, $n = 18$	Isolated lumbar extension MVC using MEDX	Lumbar extension significantly weaker in CLBP ($p < 0.05$)	Age, height and weight similar between groups
Robinson et al. (1992 ^b)	Healthy controls, $n = 12$ CLBP patients (53% having had previous surgery), $n = 16$	Isolated lumbar extension MVC using MEDX was performed and 60%MVC determined at full extension for further EMG analysis during isotonic trial (see table 3)	Absolute load used during isotonic trial was significantly lower in the CLBP group compared with the asymptomatic controls ($p < 0.05$)	10 CLBP patients had undergone previous surgery Age, height and weight similar between groups
Nelson et al. (1995)	CLBP patients, $n = 895$	Isolated lumbar extension MVC using MEDX	CLBP baseline data was compared graphically to healthy norms from (Graves et al., 1990 ^a) and shown to considerably weaker.	Patients diagnoses included non-specific CLBP, degenerative disc/arthritis disease, lumbar disc syndrome or spondylolisthesis/spondylolysis
Mooney et al. (1995)	Strip mine workers (90% reported prior LBP), $n = 197$	Isolated lumbar extension MVC using MEDX	Baseline data was compared graphically to healthy norms from (Graves et al., 1990 ^a) and shown to considerably weaker.	
Mooney et al. (1997)	Healthy controls, $n = 8$ CLBP patients, $n = 8$	Isolated lumbar extension MVC using MEDX	CLBP baseline data was compared graphically to both healthy participants in the study and healthy norms from (Graves et al., 1990 ^a) and shown to be considerably	Patients showed evidence of degenerative disc disease

Boyce et al. (2008)	Small manufacturing plant workers (53% reported LBP), $n = 20$	Isolated lumbar extension MVC using MEDX	weaker. Baseline data was compared graphically to healthy norms from (Graves et al., 1990 ³) and shown to considerably weaker.
---------------------	--	--	---

Maximal Voluntary Contraction = MVC; Chronic Low Back Pain = CLBP; Low Back Pain = LBP; Body Mass Index = BMI

If it is the case that deconditioning exists independent of previous surgery, a number of possible explanations may exist for the apparent association between ILEX deconditioning and chronic LBP: pain, anticipation of pain or pain avoidance behaviours, interfering with trunk muscle function; lack of motivation in asymptomatic participants; even deliberate malingering in some cases. Studies as early as those of McNeill et al. (1980) suggested that the reduced TEX strengths seen in symptomatic compared with asymptomatic participants are most likely explained by the participants' avoidance of either large tensions in the posterior soft tissue or large compressive force on the lumbar motion segments. This conclusion would seem reasonable being that there was an absence of studies of the lumbar extensor musculature in chronic LBP showing *in vivo* the condition of the lumbar musculature at the time of the study by McNeill et al. (1980), to corroborate with the empirical findings on function. Indeed, strength is a product of both muscular force and the moment arm about which it acts, but the measurement of strength is significantly affected by volitional exertion. The concerns of McNeill et al. (1980) were well justified in the absence of evidence specifically implicating muscular deconditioning *in vivo*. Evidence that has subsequently examined this, however, provides important information regarding the presence of specific lumbar deconditioning of the lumbar extensors in LBP. As such the next section shall detail and discuss this evidence.

2.2.4 Imaging and Histochemical Studies of the Lumbar Extensor Musculature in LBP

2.2.4.1 Considerations for Imaging and Histochemical Studies of the Lumbar Extensor Musculature

As suggested, the data on reduced ILEX function should be further corroborated with studies specifically examining the lumbar extensor musculatures condition *in vivo*. Documentation of their roles in support and stability of the lumbar spine has motivated a large body of research examining their anatomical and histochemical condition in relation to LBP. Broadly, these studies can be divided into those that have examined the gross anatomy of the lumbar musculature (using imaging study i.e. magnetic resonance imaging

[MRI] or computed tomography [CT]) and those that have examined the histochemical nature (through use of muscle biopsy), or 'microanatomy' of the lumbar musculature.

Here we will review both, yet whilst doing so consider the many factors that may affect and limit the conclusions that can be drawn from these studies. Surgery via posterior approach can result in alteration of the lumbar musculature (Weber et al., 1997) which can be lasting (Rantanen et al., 1993; Sihvonen et al., 1993). However there is evidence that only gross surgery, such as that for disc herniation, has this effect and that laminectomy and nucleotomy does not impart this damage to the musculature (Motosuneya et al., 2006). This is an important factor when considering the population examined. In some cases participants undergoing acute surgery have been examined and this presents an issue with determining whether deconditioning was present before surgery or is merely a result of surgery; indeed both may be the case (Weber et al., 1997).

Another issue that is involved in studies that have drawn bilateral (i.e. left and right) comparisons for evidence of asymmetry, or multiple vertebral level comparisons, is lack of asymptomatic controls. If deconditioning is present more on one side than the other, or more confined to a particular vertebral level, it is often considered that atrophy is local to symptoms (Hides et al., 1994). However without an asymptomatic group to compare it is impossible to say whether the asymptomatic side of symptomatic participants is normal or indeed atrophied itself, though to a lesser degree than the symptomatic side.

Additionally age significantly impacts upon muscle degeneration (Hadar et al., 1983; Lexell & Downham, 1992; Mannion et al., 2000). Research that has compared symptomatic participants to age-matched asymptomatic controls is more valuable in determining the association of deconditioning with LBP. Other considerations include the validity of semi-quantitative analysis of images (Mengiardi et al., 2006) and the value of measuring cross sectional area (CSA) as compared to muscle density or fatty infiltration in imaging studies (Hultman et al., 1993).

An issue with many of the studies examining so called 'normal' or 'asymptomatic' muscle histochemistry is that they have utilised biopsy samples from autopsy (Sirca & Kostevc, 1985) or from acute disc herniation patients undergoing surgery (Ford et al., 1983; Bagnall et al., 1984). This is justified by the assumption that short duration of spinal dysfunction would have little impact upon muscle condition (Bagnall et al., 1984), and early suggestions are that surgical procedure has little impact upon muscular condition due to asymmetric differences being unrelated to the side of herniation (Ford et al., 1983). Due to the possible association between deconditioning and the initiation of LBP, these disc herniation surgery studies may perhaps be more indicative of the typical muscle condition that predisposes LBP development if it is known that the biopsies were taken before surgery began; however this is often not reported. It is important to remember that gross surgical procedures such as those for disc herniation have themselves been shown to have an impact upon the musculature (Motosuneya et al., 2006).

As a result of these concerns this discussion focuses upon studies that have appropriately controlled for these factors (i.e. exclusion of previous surgery, control of age between groups). In addition, consideration of the potential for the presence of either deconditioning confined to a particular side or vertebral level will be compared to the potential for a general deconditioning. As with the TEX studies, those studies that have not considered such methodological factors as highlighted in this section have been summarised in the appendices¹¹ provided and appear to show inconsistent associations both for imaging and histochemical studies.

2.2.4.2 Imaging Studies of the Lumbar Musculature

Firstly, the imaging studies that have examined the gross anatomy of the lumbar musculature will be reviewed. As noted there are numerous studies on this topic that have not controlled for the potentially confounding factors of age and previous lumbar

¹¹ See appendix 7.5.2

surgery. Of those examined for this review a handful of studies (Hultman et al., 1993; Kamaz et al., 2007; Mengiardi et al., 2006; Danneels et al., 2000; Kang et al., 2007) did control for these factors and the results of them are summarised here (note that though some studies have examined the psoas also, this review focuses upon the lumbar extensors).

Several studies have examined the CSA of either the paraspinal muscles as a whole group (Kamaz et al., 2007; Danneels et al., 2000), the ES muscle group (Hultman et al., 1993; Danneels et al., 2000), and in one the quadratus lumborum (QL; which can initiate lumbar extension when bilaterally contracted; Kamaz et al., 2007). Kamaz et al. (2007) examined absolute total paraspinal muscle CSA and found significant reduction in chronic LBP participants at the lower level of L4 but not at the upper. There was however a significant reduction in CSA of the QL at the upper level. Danneels et al. (2000) found no difference in normalised ES CSA between symptomatic or asymptomatic chronic LBP participants. In addition, however, they also examined total paraspinal muscle CSA and did report significantly reduced CSA in chronic LBP participants. Comparing these results is problematic as Kamaz et al. (2007) did not normalise their values. Though Danneels et al. (2000) did not find a reduction in CSA of the ES, they did in the MF and attributed the reduction in total paraspinal CSA to the reduction in the CSA of MF.

Another study by Hultman et al. (1993) comparing participants with intermittent LBP, chronic LBP and healthy age matched controls found no difference between groups for ES CSA. They did however find a significant reduction in ES density in the chronic LBP group. This study potentially brings the value of CSA as a sole measure of deconditioning or atrophy into question and may explain some of the disparity in results of other studies. Indeed as in other physiological measures the absolute measurements of a particular variable (i.e. CSA, in a similar vein to mitochondrial volume [Luthi et al., 1986] or capillary density [Hepple et al., 1997; Green et al., 1999] that have been discussed elsewhere [Steele et al., 2012]) are often less valid than relative measures of density and the same

might apply to muscle CSA and muscle density. Muscle density may therefore be more representative of muscle atrophy as changes such as fatty infiltration may serve to maintain absolute CSA but would indicate that muscle density had indeed reduced. Yet in light of this Mengiardi et al. (2006) when comparing age matched controls found no difference in ES fat percentage. Studies examining muscle density/fat content are expanded upon below.

CSA of the MF has also been examined by two studies, both of which support a link between reduced MF CSA and chronic LBP (Kamaz et al., 2007; Danneels et al., 2000). Both Kamaz et al. (2007), and Danneels et al. (2000) demonstrated that MF CSA was significantly reduced compared with healthy age matched controls. Kamaz et al. (2007) found these results consistent at both the upper and lower level of L4 and Danneels et al. (2000) at just the lower L4 level.

When the ES has been examined for differences in muscle density or fat content between asymptomatic and symptomatic participants there have been contrasting findings. As noted, although they did not find any evidence of reduced CSA, Hultman et al. (1993) noted significant reduction in muscle density of the ES in chronic LBP participants. Danneels et al. (2000) however also found no difference in ES muscle CSA without fat between their age matched groups and so they suggested that fatty infiltration may be more closely associated with age than indicative of atrophy (Hadar et al., 1983; Lexell & Downham, 1992; Mannion et al., 2000). Mengiardi et al. (2006) when comparing age matched controls, also found no difference in longissimus fat percentage using both a quantitative and semi-quantitative analysis.

In considering the MF Danneels et al. (2000) also reported no difference in muscle CSA without fat for the MF, despite an overall reduction in MF CSA. In contrast, however, Mengiardi et al. (2006) did find a significant difference in fat percentage of the MF when considering the results of their quantitative analysis. The results of their semi-quantitative

analysis however found no difference and it seems reasonable to suggest that this lack of difference may stem from observer error.

Moderate differences between studies may perhaps be explained by the level of measurement. It has certainly been suggested and evidenced by some that atrophy of the ES or MF may be dependent upon level and side of symptoms due to denervation atrophy (Hides et al., 1994; Barker et al., 2004). However other evidence has suggested that a general atrophy at all levels and both sides may exist in chronic LBP participants compared with controls and that asymmetry is merely more pronounced in those with specific symptoms of radiculopathy (Hyun et al., 2007). Although age was controlled in one study between groups (Hides et al., 1994) and participants with previous surgery were excluded from the other two (Barker et al., 2004; Hyun et al., 2007) the lack of control of one or the other between these studies renders difficulty in concluding whether deconditioning and atrophy is level or side specific or whether it is indeed more general. This is certainly an area requiring further research, the results of which may have important implications for prevention and treatment through use of exercise particularly considering the different approaches used to address these i.e. resistance exercise or motor control training.

Although not comparing asymptomatic and symptomatic participants, one other study is worthy of mentioning which did control for the influence of age and previous surgery. Kang et al. (2007) examined CSA, both absolute and normalised to disc CSA, and used semi-quantitative analysis of fat content of the ES and MF in a group of chronic LBP participants as controls and a group of chronic LBP participants with degenerative kyphosis preparing to undergo corrective surgery. Although they were not able to compare their groups to healthy asymptomatic controls they did note that reduced CSA, both absolute and normalised, and more severe fat content, were found in the kyphosis group compared with the controls, and regression analysis showed MF atrophy to be most strongly associated. These results are interesting considering the influence of the

musculature upon spinal stability and certainly present an area of future research to examine whether the general atrophy often seen in chronic LBP is more severe in light of structural dysfunctions, and whether this is causative or instead a result of these more severe conditions.

Though disparate perhaps due to methodological issues, those studies reviewed (having excluded previous surgery and controlling for age) all suggest some form of deconditioning and atrophy, either reduced CSA, reduced density or fatty infiltrations, being present in both the ES, and the MF in of chronic LBP participants compared with asymptomatic controls (Hultman et al., 1993; Kamaz et al., 2007; Mengiardi et al., 2006; Danneels et al., 2000). Considering that both play important roles in lumbar spine stability (MacDonald et al., 2006) this is potentially evidence for a plausible role of deconditioning in LBP.

2.2.4.3 Histochemical Studies of the Lumbar Musculature

Imaging studies offer valuable insight into the gross anatomical condition of the musculature. Histochemical studies on the other hand are able to provide further detail considering individual fibre size, density and differentiation between differing fibre types, as well as identifying specific pathological changes such as presence of small angulated fibres, target/core targetoid fibres, and fibre type grouping or group atrophy (Mannion, 1999). Mannion (1999) has reviewed and highlighted a number of important findings from this area. She concludes by highlighting the difficulty of distinguishing cause and effect of fibre type characteristics (i.e. whether the observed characteristics existed prior to onset of LBP, or were a consequence of the presence of LBP). Her review also discussed fibre characteristics in relation to electromyographic (EMG) analysis of the lumbar spine musculature and this will be discussed further in the following section. Here we will further consider the findings reported by Mannion (1999) along with more recent findings. Again, studies have considered both the ES and MF (both deep and superficial).

When it has been made clear in studies that biopsies were taken before surgery then the direction of the association between deconditioning and LBP might be better identified. However, biopsies are frequently taken during surgery or this is often not clarified (Rantanen et al., 1993; Sihvonen et al., 1993; Ford et al., 1983; Zhu et al., 1989; Mannion et al., 1997^a; Fidler et al., 1975; Mattila et al., 1986; Bajek et al., 2000; Yoshihara et al., 2001). Where it is not specified it is instead prudent to assume that biopsy was taken during the operation meaning we need to treat the results from these studies with caution. One study has shown that pathological changes are present before surgery although further denervation is apparently caused by surgery as shown in biopsies taken afterward (Weber et al., 1997) which certainly suggests that deconditioning may be present before surgery is initiated and thus associated with conditions for which surgery is recommended.

Studies of the histochemical condition of the paraspinal muscles in symptomatic chronic LBP participants that have not undergone surgery suggest the presence of fibre atrophy, pathological changes, and fibre type ratio alteration (Mannion et al., 2000; Zhao et al., 2000). Neither of these cited studies, however, included asymptomatic controls. Zhao et al. (2000) conducted bilateral comparisons and suggested that different findings between sides were affected by location of herniation, and that differences existed among those with central, bilateral and unilateral pain. Prospective evidence has suggested that herniation can cause change in muscle activity, which might cause denervation atrophy (Haig et al., 1993). Again however the absence of an asymptomatic control group renders the same difficulty as in other studies (Hides et al., 1994; Barker et al., 2004) when drawing conclusions (i.e. it is not known if the side without herniation was atrophied also).

Only one study has been conducted in the absence of the potential confounding influence of surgery, has controlled for the confounding effects of age, and also included a matched asymptomatic control group (Crossman et al., 2004). Crossman et al. (2004) reported no difference in fibre size or fibre ratios between participant groups and that both had a predominance of type I fibres. This is in contrast to Mannion's (1999) earlier conclusions

that symptomatic participants have a higher proportion of type IIX fibres. However Crossman et al. (2004) did not note the specific location of their biopsy sample and thus it is not clear whether these results refer to the ES, MF or the paraspinal musculature as a whole.

2.2.4.4 Summary of Imaging and Histochemical Studies of the Lumbar Extensor Musculature

Although evidence suggests that deconditioning is indeed present in some form in symptomatic participants there is considerable disparity in methodologies in both imaging and histochemical studies. Data from imaging studies appear more consistent in their findings of some form of atrophy (Hultman et al., 1993; Kamaz et al., 2007; Mengiardi et al., 2006; Danneels et al., 2000) as opposed to those from histochemical studies; however only one histochemical study has controlled for previous surgery and age (Crossman et al., 2004). Indeed although general deconditioning may be present in LBP, the findings of Crossman et al. (2004) suggest that dominance of an adverse fibre type is perhaps not. Table 2 summarises these studies.

Table 2. Summary of imaging and histochemical studies of the lumbar extensor musculature in LBP

Reference	Participants	Testing	Results	Comments
Imaging Studies				
Hultman et al.(1993)	Healthy controls without history of LBP, $n = 24$	CSA and density of erector spinae using CT at L3 level	Muscle density was significantly lower in CLBP patients compared to both other groups ($p < 0.05$)	Those with previous lumbar surgery were excluded
	Patients with intermittent LBP, $n = 40$		CSA did not significantly differ between groups	Age, height, body mass and body composition similar between groups
	CLBP patients, $n = 21$			
Kamaz et al. (2007)	Healthy controls without LBP or leg pain, $n = 34$	CSA of total paraspinal, multifidus, quadratus lumborum, psoas and gluteus maximus muscles using CT at L4 upper and lower plates	CSA was significantly reduced in only paraspinal and multifidus at the lower plate in CLBP ($p < 0.01$)	Those with previous lumbar surgery were excluded
	CLBP patients, $n = 36$		CSA was significantly reduced in only multifidus, psoas and quadratus lumborum at the upper plate in CLBP ($p = 0.05$)	Age and BMI similar in both groups.
			No significant differences between CSA of gluteus maximus	
Mengiardi et al. (2006)	Healthy controls without history of LBP in previous 2 years, $n = 25$	CSA of multifidus and longissimus fat content and semi-quantitative grading using 5 classification system using MRI at L4-L5 level	CLBP patients showed significantly greater fat content in the multifidus ($p < 0.05$)	Those with previous lumbar surgery were excluded
	CLBP patients, $n = 25$		No difference found using semi-quantitative system	Age, sex and BMI matched between participant groups
Danneels et al. (2000)	Healthy controls without history of previous LBP, $n = 23$	Total CSA and muscle CSA of total paraspinal, erector spinae, multifidus and psoas muscles using CT at upper L3, and upper and lower L4 normalised	Total CSA of paraspinal and multifidus muscles significantly smaller at lower L4 in CLBP ($p < 0.05$)	Those with previous lumbar surgery were excluded in addition to those who had participated in training for the lower back muscles in the previous 3 months
	CLBP patients, $n = 32$		No significant difference for erector spinae or psoas	Age, height, weight and activity similar between groups
Kang et al. (2007)	CLBP patients with lumbar degenerative kyphosis undergoing corrective surgery, $n = 54$	CSA and muscle to disc CSA ratio of psoas, erector spinae and multifidus was assessed at L4/L5 level and fatty infiltration of psoas, erector spinae and multifidus assessed at L3/L4 using three grade	CSA and muscle to disc CSA ratios for all muscles were significantly lower in the lumbar degenerative kyphosis group compared with controls ($p < 0.001$) with regression analysis showing multifidus wasting	No healthy control group for comparisons
	CLBP control patients, $n = 54$			Those with previous lumbar surgery were excluded from CLBP control group

classification using MRI

to be most strongly associated ($p < 0.001$)

Severe fatty infiltration was significantly more common in lumbar degenerative kyphosis compared to CLBP controls ($p < 0.05$)

Age and sex matched between groups and symptom durations were similar

Body mass and BMI was significantly higher in CLBP controls

No difference in degenerative changes (degenerative disc disease, herniation's, stenosis or spondylolithesis) between groups

Histochemical Studies

Crossman et al. (2004)

Healthy controls without LBP lasting >3 days in previous 12 months, $n = 32$

Percutaneous biopsy of paraspinal muscle (specific location not noted) for fibre CSAs and fibre typing.

No significant differences between groups for any fibre histochemical comparisons

Those with previous lumbar surgery were excluded

CLBP patients, $n = 35$

Age, gender and all anthropometric characteristics similar between groups

Cross Sectional Area = CSA; Computed Tomography = CT; Magnetic Resonance Imaging = MRI; Chronic Low Back Pain = CLBP; Low Back Pain = LBP; Body Mass Index = BMI

Crossman et al. (2004) also suggest that the differences in functional tests between asymptomatic and symptomatic participants' strength/endurance discussed in the previous section may therefore be due to the influences of psychological disturbance or motivation. However, we should consider that it is not only fibre type distribution that influences fatigue resistance but also capillary density, enzymatic activities and associated metabolic processes (Mannion, 1999; Steele et al., 2012). So it is unsurprising that there is not a distinct relationship between this single variable and its associated end effect. Mannion (1999) highlights that, because functional tests (i.e. strength/endurance) can be confounded by psychological disturbance, EMG should be employed to circumvent this and record more objective indices of muscle activation and fatigue. Indeed this measure might be considered to account for the many factors influencing fatigue due to its ability to accurately predict it (Roy et al., 1989) and that it also has a close association with physiological indicators of fatigue (Boissou et al., 1989; Vestergaard et al., 1992; Laurent et al., 1993). Cooper et al. (1993) have shown that greater EMG amplitude increases occur during a test to fatigue in symptomatic participants (both surgical and non-surgical, suggesting a similarity underlying the two groups) compared with asymptomatic participants and suggested that it indicated an increased central drive secondary to muscle wasting or denervation. Thus EMG and other activation studies therefore may provide further insight into the deconditioning hypothesis and LBP.

Evidence suggests reduced strength/endurance in symptomatic participants which is further corroborated with *in vivo* evidence of muscular deconditioning being present. Further, and in consideration of the aforementioned concerns with participant effort in functional tests, the following section will complete the triumvirate of areas covered in examining deconditioning of the lumbar extensor musculature in LBP by reviewing studies that have employed EMG to assess fatigability.

2.2.5 Electromyography Studies of Fatigue in the Lumbar Extensor Musculature in

LBP

2.2.5.1 Considerations for Electromyographic Fatigue Analysis of the Lumbar Extensor Musculature

In consideration of the effect that deconditioning, and thus fatigability, may have on LBP, EMG has been used to attempt to control for influence of psychological disturbance or participant motivation on functional measures of endurance (Mannion, 1999). Thus the information presented by these studies is also useful in examining the deconditioning hypothesis by corroborating evidence from the prior two sections which may support a link between lumbar extensor deconditioning and LBP. EMG can be used to interpret muscle activation and muscle force (De Luca, 1997) but can also be used to more objectively demonstrate fatigability (Mannion, 1999; Roy et al., 1989). EMG is limited in many regards by such confounding factors as crosstalk (readings from synergist muscles), depth of active motor units from surface electrode, amplitude related to motor units and muscle fibre-types, variable firing rates, muscle-fibre length, velocity and contraction type (De Luca & Merletti, 1988; Wakeling et al., 2001; Roman-Liu & Tokarski, 2002; Farina et al., 2004; Semmler et al., 2007; Roberts & Gabaldon, 2008). Cross talk is of particular issue when differentiating specific lumbar extensor musculature (Stokes et al., 2003). However, in considering power spectrum analysis of rate of change in EMG spectral variables (De Luca, 1993; i.e. root mean square amplitude, mean, median or mode frequency slopes, initial frequencies etc.) for determining fatigability these might perhaps not be so confounding an issue as they would presumably remain constant systematic errors while such EMG parameters would change with fatigue.

When looking at LBP populations we should consider that the MVC-normalised EMG signal amplitude measured may perhaps be influenced by insincere effort (Pitcher et al., 2008; Watson et al., 1997). Roy et al. (1995) however, have shown that EMG measures of fatigability provide accurate classification independently of MVC, suggesting their greater

objective power in discriminating between symptomatic and asymptomatic groups compared to simply measuring relative activation levels.

Although it seems EMG measures of fatigability are more valid, a point must be considered when interpreting their results; that is, whether participants performed exercise to momentary muscular failure (MMF; i.e. maximal intensity of effort; Carpinelli et al., 2004; Fisher et al., 2011; Steele, 2013^b). These studies should not surprisingly show a difference in fatigue indices from start to end of exercise performance within all groups participating in testing, but presumably would show no difference in between-group comparisons as both will be maximally fatigued. Change in fatigue indices over a fixed number of repetitions or time (i.e. as an *absolute* measure) would instead be the most appropriate means of detecting fatigue-related differences between symptomatic and asymptomatic groups, and, considering the issue with normalising to MVC in LBP participants, should preferably utilise an absolute load. In some studies the absolute load utilised has been the participant's torso mass during TEX. Thus an important consideration for between-group comparisons of fatigue during TEX is whether body or torso mass was similar.

Geisser et al. (2005) have conducted a meta-analysis of the use of trunk surface EMG comparing asymptomatic and symptomatic participants and comment that EMG recordings from non-maximal tasks are likely to be more reliable than those involving maximal exertions. However, we should remember that both absolute and relative amplitude levels will be subject to the aforementioned limitations of EMG including an insincere effort, whereas fatigue may not be. EMG measures of fatigability should objectively quantify fatigue independently of MVC (Roy et al., 1995) where a significant change in fatigue is unlikely to be seen if insincere effort is put forth. Geisser et al. (2008) reported an effect size of zero for EMG measures of fatigability during isometric trunk exertions, suggesting no difference between symptomatic and asymptomatic participants. However, a difficulty lies in interpreting these results partly due to the methodological

differences of studies included but also because Geisser et al. (2008) do not clarify EMG locations and whether extensor or flexor musculature, or a combination of the both, was being examined. Nor do they comment in more detail on the intensity of effort of the activity (i.e. whether it was performed to MMF or to an absolute time/number of repetitions). Their meta-analysis included 7 studies (Kankaanpaa et al., 1998^a; Kankaanpaa et al., 1998^b; Paasuke et al., 2002; Suter et al., 2001; Robinson et al., 1992^b; Roy et al., 1989; Mayer et al., 1989^a; Peach & McGill, 1998) examining EMG measures of fatigability in LBP; however, a number of studies also examining fatigue indices that were present at the time of its publication were not included (Roy et al., 1995; Roy et al., 1990; Biedermann et al., 1991; Klein et al., 1991; Mannion et al., 1997^b). Being that EMG measures of fatigability are more valid and applicable to our present discussion of the deconditioning hypothesis we will further examine the studies analysed by Geisser et al. (2008) along with those not included in their analysis, as well as further studies that have been conducted more recently (Lariviere et al., 2010^a; Humphrey et al., 2005; Suuden et al., 2008; Lariviere et al., 2011; Da Silva et al., 2005).

For sake of clarity in this review, although numerous methods of analysing the EMG signal for determination of fatigability exist between studies, here these methods are collectively referred to as EMG 'fatigue indices', as a critical comparison of the specific methods of analysis is beyond the scope of this review (Lariviere et al., 2008). Due to the difficulty of cross talk between the paraspinal musculature when using surface EMG (Stokes et al., 2003), we do not attempt to differentiate between, for example, the ES or MF, and instead consider the studies reviewed to offer information regarding the lumbar extensor musculature as a whole. Being that, as previously noted, surgery can have considerable confounding effects upon the lumbar extensor musculature (Weber et al., 1997; Rantanen et al., 1993; Sihvonen et al., 1993; Motosuneya et al., 2006), we have focused in this section upon those studies which have controlled for this (Crossman et al., 2004; Da Silva et al., 2005, Roy et al., 1989; Mayer et al., 1989^a; Humphrey et al., 2005). Again as with previous sections those studies excluded from discussion (in this case those not

controlling for surgery or those which have had participants perform exercise to MMF) have been summarised within the appendices¹².

2.2.5.2 Fatigability Studies of the Lumbar Musculature

The studies reviewed utilising measurements of EMG fatigue indices have examined differences between asymptomatic and symptomatic participants using different methods. Some have used both discriminant analysis and regression to identify whether such measures can successfully classify participants, and others have drawn simpler between group comparisons.

Roy and colleagues have performed several studies examining fatigue indices in LBP, one of which controlled for both factors noted (Roy et al., 1989). They examined fatigue indices during 60 second standing isometric TEX contractions at 40%, 60% and 80% MVC. Discriminant analysis correctly classified between asymptomatic controls and symptomatic chronic LBP participants at 40% MVC (92% controls, 82% chronic LBP) and 80% MVC (84% controls, 91% chronic LBP), however results were less favourable at 60% MVC (67% controls, 75% chronic LBP; a later study by Peach & McGill (1998) clarifies this anomaly though it should be noted they do not note whether those with previous surgery were excluded). This study also looked at two level analysis (Lumbar level and %MVC level) finding that fatigue indices at L5 for 80% MVC showed the most favourable classification (75% controls, 75% chronic LBP).

In an early study by Mayer et al. (1989^a) participants performed an isometric TEX hold using a roman chair in the same manner as the Biering-Sorensen test. Participants performed a series of 10 holds for 15 seconds each with a rest period of 10 seconds between holds. Between group comparisons of fatigue indices for both the first 5 holds, as well as the full 10, demonstrated significantly greater fatigue in the symptomatic chronic LBP group than in the asymptomatic controls before completion of an intensive

¹² See appendix 7.5.3

rehabilitation program. After the program the difference between groups was reduced and still significant for the 10 holds, yet there was no significant difference when data for 5 trials were compared.

Humphrey et al. (2005) considered a range of fatigue indices calculated from power spectrum analyses during a back lift test. They reported significant differences in fatigue indices between chronic LBP participants and controls. In addition they reported that logistic regression showed high sensitivity and specificity in classifying chronic LBP participants. However, although the variables considered could discriminate between symptomatic chronic LBP participants and asymptomatic controls there were varying degrees of accuracy. They noted that those variables that could potentially be affected by load (peak amplitude, median frequency) were less accurate as predictors; however, load independent variables (such as initial median frequency and half width) offered a higher degree of accuracy. Humphrey et al. (2005) also included a group of participants with a past history of LBP. No variables were able to discriminate these from either the chronic LBP participants or the controls though this was suggested to be due to the comparatively small sample size for this group (healthy controls $n = 175$; chronic LBP participants $n = 145$; past history participants $n = 30$).

A later study by Da Silva et al. (2005) however, offers contrasting results. They found no significant difference between asymptomatic controls or symptomatic chronic LBP participants in fatigue indices between groups for 60 second contractions at 50% MVC for the Biering-Sorenson test, a standing extension test, and also a semi-crouching back lift test. It is unclear as to the reason for this contrasting finding; however, Da Silva et al. (2005) suggest that the chronic LBP group studied may not have been sufficiently impaired to demonstrate a difference based upon the low results from the Oswestry disability questionnaire (~12%).

Another study by Crossman et al. (2004) has also reported no difference in fatigue indices between healthy asymptomatic and symptomatic chronic LBP participants during standing TEX using 60%MVC for 60 seconds and also during the Biering-Sorenson test. Crossman et al. (2004) comment that this may perhaps not be surprising due to the lack of differences in histochemical analysis of fibre typing in their participants. However they also note concerns regarding the loads used by chronic LBP during the 60%MVC TEX test in particular, speculating that these participants may not have given a sincere MVC and thus they may have been using <60%MVC during this test. As noted earlier MVCs have been evidenced to be affected by this (Pitcher et al., 2008; Watson et al., 1997) and as a result we noted that the use of an absolute load may be of greater validity in determining differences in fatigability. Crossman et al. (2004) did also have participants perform the Biering-Sorenson TEX test, also reporting similar body mass between groups, which suggest that the absolute loading between groups for this test was similar. However this test was performed to MMF and so it is again unsurprising that no differences in fatigue were found. Yet, chronic LBP participants did demonstrate significantly lower endurance times and so assuming they did perform the test to MMF (and that also healthy controls did) the lower endurance time might indicate greater fatigability. However it again must be noted that this is a test of TEX and so the endurance time is not specifically indicative of the lumbar extensors.

Unfortunately, considering the potentially confounding effect of relative load being influenced by insincere MVCs in chronic LBP participants, we are left with only the results of Mayer et al. (1989^a) which do suggest greater fatigability in chronic LBP participants. It is difficult to discern whether any other factors may have affected the results between studies apparently supporting the presence of deconditioning through EMG fatigue indices (Humphrey et al., 2005; Roy et al., 1989; Mayer et al., 1989^a) and those suggesting it is not present (Crossman et al., 2004; Da Silva et al., 2005). All have used a range of electrode placement sites (T10/L1/L2/L3/L4/L5), many in different combinations, a range of tests (standing TEX, prone TEX, back lift test), both relative and absolute loads as

noted, and also a range of test timings (30 seconds, 60 seconds and 10 repetitions of 15 seconds); as such it is unclear as to what effect these variables may have upon the study's findings.

2.2.5.3 Summary of Electromyography Studies of Fatigue of the Lumbar Extensor Musculature

In summary, of the studies reviewed, it appears that objective measures of fatigability show contrasting results. Those controlling for previous surgery and using standardised timed protocols show some evidence in support (Humphrey et al., 2005; Roy et al., 1989; Mayer et al., 1989^a) and some against (Crossman et al., 2004; Da Silva et al., 2005) the presence of deconditioning. One study that has also controlled for the potentially influencing factor of sincere effort by chronic LBP participants does, however, suggest the presence of some degree of deconditioning (Mayer et al., 1989^a). Table 3 summarises these studies.

Table 3. Summary of studies testing fatigability with EMG of the lumbar extensor musculature in LBP

Reference	Participants	Testing	Results	Comments
Crossman et al (2004)	Healthy controls without lasting >3 days in previous 12 months, <i>n</i> = 32	EMG recorded bilaterally from lumbar paraspinal muscles at L4-L5 level during standing isometric trunk extension for 60 seconds at 60%MVC and during the Biering-Sorensen test	EMG fatigue indices were similar between groups for the Biering-Sorensen test and also the 60%MVC test	Those with previous lumbar surgery were excluded
	CLBP patients, <i>n</i> = 35			Age, gender and all anthropometric characteristics similar between groups
Da Silva et al. (2005)	Healthy controls without history of LBP in previous year, <i>n</i> = 15	EMG recorded bilaterally from lumbar paraspinal muscles at T10, L1, L3, and L5 levels during standing trunk extension and back lift at 50%MVC for 60 seconds, and during Biering-Sorensen test for 60 seconds	No difference in EMG fatigue indices between groups	Those with previous lumbar surgery were excluded
	CLBP patients, <i>n</i> = 13			Age, height and weight similar between groups
Roy et al. (1989)	Healthy controls, <i>n</i> = 12	EMG recorded bilaterally from lumbar paraspinal muscles at L1, L2 and L5 levels during standing isometric trunk extension for 60 seconds at 40%MVC, 60%MVC and 80%MVC	Discriminant analysis of EMG fatigue indices successfully classified 92% controls, 82% CLBP at 40%MVC, 67% controls, 75% CLBP at 60%MVC and 84% controls, 91% CLBP at 80% MVC	Those with previous lumbar surgery were excluded
	CLBP patients, <i>n</i> = 12			Age, height and weight similar between groups
Mayer et al. (1989 ^a)	Healthy controls, <i>n</i> = 11	EMG recorded bilaterally from lumbar paraspinal muscles at L3 level 3cm from midline during 10 isometric trunk extension holds on a roman chair lasting 15 seconds each and with 10 seconds rest between each hold	EMG indices of fatigue showed significantly greater fatigue in the CLBP group compared to controls (<i>p</i> < 0.01)	Those with previous lumbar surgery at level of EMG placement were excluded
	CLBP patients, <i>n</i> = 10			Age and torso weight similar between groups

Maximal Voluntary Contraction = MVC; Electromyography = EMG; Chronic Low Back Pain = CLBP; Low Back Pain = LBP; Body Mass Index = BMI

Thus far it has been evidenced that there may be an association between measures of, and variables associated with, lumbar extensor deconditioning (i.e. reduced strength/endurance testing of lumbar musculature, deconditioning shown by imaging and histochemical examination of the lumbar musculature, and increased fatigability of the lumbar musculature shown by EMG fatigue indices) and LBP, hence providing support towards the deconditioning hypothesis. Theoretically, muscular deconditioning could lead to instability and altered joint biomechanics and thus result in injuries (either single macro-trauma or cumulative micro-trauma) which may instigate pain causing mechanisms. Therefore we might expect that the presence of such deconditioning, whilst being a consistent association with CLBP, might also predict the development of LBP or incidence of low back injury in initially asymptomatic individuals also. Prospective studies have examined whether this is in fact the case and the following section will discuss the evidence implicating deconditioning's effect upon injury and development of LBP.

2.2.6 Prospective Studies of Lumbar Extensor Deconditioning in LBP

A concern with cross-sectional studies is that causation cannot be logically determined from association (it should also be noted that a lack of association does not necessarily imply a cause and effect relationship does not exist). Despite a consistent association being one of the criteria for causation as determined by Austin Bradford Hill (1965), and the consistency of some degree of deconditioning with chronic LBP, in addition to biological plausibility of which there is evidence implicating deconditioning, it still cannot solely be taken as evidence for a causative relationship, nor the direction of that causation. Prospective studies provide clearer indication for a temporal relationship between variables and allow us to consider whether the potential plausibility for deconditioning to actually lead to LBP can be evidenced.

Although in previous sections we have been selective over those studies discussed based upon methodological considerations highlighted, this section considers a more liberal range of literature. The reason for this is due to the relative paucity of prospective studies

that have appropriately controlled for the factors previously highlighted in this review. Thus the studies reviewed in this section should be considered tentatively and it is noted that further research is required to definitively test the deconditioning hypothesis and the presence of a prospective relationship between deconditioning and LBP.

2.2.6.1 Prospective Evidence from Strength & Endurance in LBP

Biering-Sorensen et al. (1984) found that weak TEX was a predictive residual sign of recurrent LBP or chronic LBP over a 1 year follow up, although did not significantly predict first time occurrence. A study by Leino et al. (1987) indicated that there was little prognostic value of tests of dynamic TEX in predicting LBP over a 10 year period but suggested instead an effect of the latter upon the former (i.e. symptoms, or degree of symptoms at baseline, had prognostic value in predicting reduced trunk muscle function at follow up). Disappointingly, however, Leino et al. (1987) omitted dynamic TEX tests from their follow up testing. Initial testing consisted of prone dynamic TEX efforts while follow-up data are reported for standing isometric TEX efforts. This presents difficulty in interpreting the effect of LBP presence at base-line affecting TEX muscle function at follow up as it is a case of comparing different tests to identify change (Mooney & Anderson, 1994). This makes the conclusions questionable. The dynamic tests consisted of the number of repetitions performed over 30 seconds which might be considered more specifically a test of the ability to complete TEX movement quickly, not TEX strength/endurance *per se*. The isometric test on the other hand was indeed a TEX MVC and thus a measure of TEX strength. The data from Leino et al. (1987) compare two entirely different tests with no clear conclusions being evident. It would seem that there was little difference in relative risk of LBP development when low and high performers in dynamic extension were followed up. Contrastingly Rissanen et al. (2002), utilising the same dynamic test reported significant prediction of back disorder disability over an average 12 year follow-up. However, such dynamic TEX testing does not really offer an appropriate presentation of muscle function (Bemben et al., 1988; Mooney, 1992; Murray, 1986) and so interpreting the predictive results from this in light of the deconditioning

hypothesis is questionable. It should also be pointed out that again that tests of TEX are not specifically indicative of lumbar extensor muscle function.

A study by Newton et al. (1993) using a more consistent study design of dynamic isokinetic TEX testing (i.e. utilising the same test both at baseline and follow up) in prospective evaluation of LBP development, however, also suggested that it held no predictive value. Despite this, prospective implementation of the same battery of isokinetic tests as a pre-employment fitness evaluation in order to place workers in appropriate job areas has been demonstrated that it can significantly reduce injury rates (Reimer et al., 1994). This suggests a potential connection between physical function and task demands in predicting injury and perhaps explains the lack of predictive value in these tests when this is not considered (Newton et al., 1993).

Batti'e et al. (1989^a) reported that greater strength was actually a risk factor for report of back problems over a 4 year period. However, closer inspection of their results shows that this was only significant for arm and leg lift strength and that torso lift (TEX) was not significant. When their data were adjusted for age there were no significant correlations. Another prospective study has reported that a reduced trunk extension/flexion strength ratio is a significant risk factor for development of LBP over a 5 year period (Lee et al., 1999). That extensor deconditioning may be more significant than flexor deconditioning in those with LBP has been highlighted in previous research (Mayer et al., 1995; McNeil et al., 1980; Addison & Schultz, 1980; Parkkola et al., 1993; Kamaz et al., 2007; Mooney et al., 1997; Bouche et al., 2011; Danneels et al., 2000) and it thus is interesting that a greater relative deconditioning of the extensors is shown to be predictive of future LBP. Kujala et al. (1996) on the other hand suggested that neither isometric nor dynamic TEX performance were predictive of first time LBP in addition to strength ratio being unrelated in their sample group. However, their results did indicate a significant effect of musculoskeletal loading as well as reporting that taller participants (who may experience greater loading due to a greater external TEX moment) were more likely to develop LBP.

Thus their results are somewhat supportive of the concepts conveyed by Reimer et al. (1994), and also Chaffin et al. (1978) and Keyserling et al. (1980) using the same test battery as Batti'e et al. (1989^a), in that strength relative to physical demands is important. So although the population studied did not differ in their initial strength, those who engaged in heavier loading were weaker relative to their loading demands (Kujala et al., 1996).

Associations between weak TEX and LBP have also been reported in younger populations (Salminen et al., 1992; Balague et al., 1993; Salminen et al., 1995). The studies of Salminen et al. (1992; 1995) involved a 3 year follow up and showed weak TEX associated with LBP at baseline and follow up. Despite this there was no predictive validity of TEX in development of LBP at follow-up. Studies have, however, also examined adolescents, showing prospective associations between TEX weakness and development of LBP (Lee et al., 1999; Sjolie et al., 2001).

TEX endurance has also been used in prospective studies. Poor TEX endurance has been identified as a risk factor for LBP incidence in some studies (Biering-Sorensen, 1984; Luoto et al., 1995; Sjolie & Ljunggren, 2001). However, one study's findings indicate that it has no use in predicting future LBP (Gibbons et al., 1997). Gibbons et al. (1997) note however that the difference in results between theirs and previous studies may be due to type II error. Their sample size ($n = 43$) for follow up in incidence of LBP after initial testing was much lower than the sample used by Biering-Sorensen (1984; $n = 982$), and also the samples used by Luoto et al. (1995; $n = 126$) and Sjolie & Ljunggren (2001; $n = 86$) which might suggest that their data would be more likely to present a type II error (i.e. a failure to reject the null hypothesis) from a lack of statistical power through low sample size. Thus, the larger and more numerous studies do indicate the predictive potential of low TEX endurance in development of LBP.

Adams et al. (1999) conducted a large prospective study examining physical factors; including TEX endurance and back lift test MVC and examined EMG fatigue indices over 20 seconds. Their results suggest back lift strength was not predictive of LBP, but TEX endurance time was. An earlier study by Mostardi et al. (1992) that demonstrated no predictive value of strength also performed a back lift test. In spite of this, another larger study ($n = 1652$) has also employed the same back lift method amongst other fitness measures and found that there was significant predictive value between those with the lowest, middle and best strength and fitness; the least fit sustaining the greatest proportion of low back injuries and the most fit sustaining the least (Cady et al., 1979).

Where many previous studies have used less valid measurements of lumbar function (i.e. TEX testing), another prospective study by Mooney et al. (1996) examined low back injury rates and their relationship to ILEX strength. One hundred and fifty two shipyard workers were tested for ILEX strength and followed up for 2 years. In this period 9% ($n = 14$) reported low back injuries; however only 2 of these had below normal ILEX strength. These injury rates (9%), however, are considerably less than those reported for many other US industries (Guo et al., 1999). The majority of the workers tested in the study by Mooney et al. (1996) had normal ILEX strength. Thus the relatively low rates of injury for the participant sample as a whole actually suggest that normal strength may be protective and that the injuries that were sustained were potentially outliers. Indeed, of the injuries reported the highest incidence was within the heavy work categories and thus these injuries may have represented accidents during heavy work (Bigos et al., 1986) whereby task demands exceeded physical function (Reimer et al., 1994; Kujala et al., 1996; Chaffin et al., 1978; Keyserling et al., 1980); however, no further detail was reported on the nature of the sustained injuries.

2.2.6.2 Prospective Evidence from MRI & EMG in LBP

Although most prospective studies have examined the role of deconditioning from a perspective of functional tests of strength and endurance, there have been others that

have examined imaging tests of the lumbar extensor musculature as well as EMG fatigue indices of the lumbar extensors. Gibbons et al. (1997), using MRI, examined CSAs, proton density weighted signals and T2-weighted signal intensities of the ES, QL and psoas major prospectively yet found no significant predictive value from any of these variables. Participants who suffered from LBP during the follow up period did show slightly higher signal intensities which might be indicative of greater fatty infiltration and thus muscular deconditioning. The fact that these were not found to be significant may be a result of a type II error again due to the sample size used (n=128). Although similar to the higher sample sizes in studies of TEX endurance, the authors are not aware of any other prospective imaging studies and so, unlike the endurance tests, this cannot be compared and confirmed.

Adams et al. (1999) also utilised EMG fatigue indices in their study yet found that there was no predictive value over 3 years follow-up. Another study by Mannion et al. (1997^b), however, reported prospective data for 200 young nurses who had never before suffered from serious LBP. EMG fatigue indices were recorded at baseline and followed up for 12 months. The result showed that greater fatigability significantly predicted development of first time LBP. Stevenson et al. (2001) reported on a variety of variables included in a predictive model of LBP over a 2 year period, including EMG fatigue indices in the final model which were significantly predictive of LBP occurrence in the previous 6 months. Finally a study by Heydari et al. (2010) has also examined EMG fatigue indices prospectively in 105 participants with no previous history of LBP. They also reported that greater fatigability was predictive of subjects self-rating of LBP at 2 year follow-up.

2.2.6.3 Summary of Prospective Studies

It seems that a number of prospective studies are suggestive of deconditioning as potentially etiological within development of LBP (Biering-Sorensen, 1984; Gibbons et al., 1997; Lee et al., 1999; Salminen et al., 1995; Sjolie & Ljunggren, 2001). These studies have predominantly employed methods examining TEX strength, endurance and trunk

extension/flexion ratios and so, as highlighted in the discussion of studies examining strength and endurance, it must be considered that there are limitations to these methods. However, one study has prospectively examined ILEX, yet, due to the limitations of this study, and depending on perspective, its data could be interpreted either in support of or against lumbar extensor deconditioning as being causative in LBP development (Mooney et al., 1996). Evidence from other methods of examining deconditioning is contrasting. MRI shows no predictive value (Gibbons et al., 1997); however, as discussed there is a lack of other imaging studies to compare this result to. In contrast, it appears that EMG fatigue indices may be predictive of LBP development (Mannion et al., 1997^b; Stevenson et al., 2001; Heydari et al., 2010). Thus, though disparate there is certainly some prospective evidence to support the deconditioning hypothesis. Table 4 summarises these studies.

Table 4. Summary of prospective studies of lumbar extensor musculature deconditioning in LBP

Reference	Participants	Testing	Results	Comments
Biering-Sorenson (1984)	Men aged between 30, 40, 50, and 60 years old, <i>n</i> = 449 Women aged between 30, 40, 50, and 60 years old	Biering-Sorensen test conducted at baseline 1 year follow-up with questionnaire concerning first time occurrence, recurrence or persistence of LBP	First time occurrence was significantly associated with low endurance time	
Leino et al. (1987)	<i>Baseline participants</i> Participants with "Good" low back status, <i>n</i> = 578 Participants with "Intermediate" low back status, <i>n</i> = 260 Participants with "Bad" low back status, <i>n</i> = 64 <i>Follow-up participants</i> Participants with "Good" low back status, <i>n</i> = 239 Participants with "Intermediate" low back status, <i>n</i> = 203 Participants with "Bad" low back status, <i>n</i> = 210	Standing dynamic trunk extension/flexion maximum repetitions performed over 30 seconds with buttock and thighs against a supporting plate and ankles tied by a belt conducted at baseline Standing isometric trunk extension/flexion MVC with buttock and thighs against a supporting plate and ankles tied by a belt conducted at 10 year follow-up in addition to questionnaire and assessment of low back symptoms and status	Trunk strength was not predictive of low back symptoms or status at follow up.	
Luoto et al. (1995)	Healthy participants without history of LBP in previous year at baseline, <i>n</i> = 167	Biering-Sorensen test and questionnaire regarding previous and present LBP conducted at baseline 75% of participants were available for follow-up at 1 year with the same questionnaire, <i>n</i> = 126	Endurance time was significantly associated with first time occurrence of LBP when adjusted for age, sex and occupation ($p < 0.05$) Endurance time broken into tertiles (poor, medium, good) showed a non-linear dose-response relationship with first time occurrence of LBP ($p < 0.04$) Relative odds ratio compared to 'good' for 'medium' and 'poor' were 1.4 (95% CI 0.4 - 4.2) and 3.4 (95% CI 1.2 - 10.0) respectively	

Gibbons et al. (1997)	Healthy participants without history of LBP in previous year at baseline, $n = 43$	Isokinetic back lift MVC, psychophysical back lift test, Biering-Sorensen test, CSA, proton-density weighted signal, and T2-weighted signal of erector spinae, quadratus lumborum, psoas major and total paraspinal muscle using MRI, and interview regarding previous and present LBP conducted at baseline Interviews regarding LBP were conducted at 1 year follow-up	Neither back lift, psychophysical back lift or endurance time differed between those with and without LBP at follow-up, nor were they associated with frequency of LBP at follow-up Neither CSA, proton-density weighted signal, or T2-weighted signal differed between those with and without LBP at follow-up, however, total paraspinal CSA, and proton-density weighted signal and T2-weighted signal of erector spinae, quadratus lumborum, psoas major were significantly associated with frequency of LBP at follow-up ($p < 0.05$)	
Mannion et al. (1997 ^b)	Healthy nurses without history of LBP, $n = 200$	EMG recorded bilaterally from lumbar paraspinal muscles at T10 and L3 level 3-4cm from midline during Biering-Sorensen test and maintenance of 80%MVC for 28 seconds at baseline Postal questionnaire regarding LBP conducted at 1 year follow-up	13% developed serious first time LBP during the follow-up period EMG indices of fatigue during Biering-Sorensen showed greater fatigue was significantly associated with development of first time LBP at follow-up ($p < 0.05$) however endurance time was not associated with first time LBP	
Rissanen et al. (2002)	Participants from the Mini-Finland Health Survey, $n = 535$	Dynamic trunk extension/flexion maximum repetitions performed over 30 seconds with buttock and thighs against a supporting plate and ankles tied by a belt conducted at baseline Average 12 year follow-up to time until retirement due to work disablement, death or end of observation period for primary diagnosis as cause of work disability	At follow-up of 56 incident cases 15 were due to back disorders Adjusted relative risks in multiple models showed trunk extension performance significantly predicted back disorder disability risk ($p = 0.04 - 0.002$)	
Newton et al. (1993)	Healthy participants without history of LBP, $n = 70$	Isokinetic trunk extension, flexion, rotation, and back lift MVC and psychophysical lift conducted at	23% developed LBP during the follow-up period, yet at least 6 months after initial assessment in all	Those with previous lumbar surgery were excluded

		baseline	cases	
		1 year follow-up with questionnaire concerning first time occurrence, recurrence or persistence of LBP	None of the isokinetic measures differed between those who did and those who did not develop LBP	
Reimer et al. (1994)	Healthy prospective order selector employees for 1989, $n = 122$	Dynamic lift capacity, isokinetic trunk extension, flexion, rotation, and back lift MVC and psychophysical lift conducted at baseline to determine placement in employment as an order selector in a warehouse grocery distributor	After implementation of prospective evaluation for employment placement in 1989, incidence of low back injuries were significantly reduced by 32% in 1990 and 41% in 1991 ($p < 0.001$)	
	Healthy prospective order selector employees for 1990, $n = 122$			
	Healthy prospective order selector employees for 1991, $n = 122$	2 year follow-up with questionnaire concerning first time occurrence, recurrence or persistence of LBP		
Batt'ie et al. (1989 ^a)	Employees working for a large aircraft manufacturer ($n = 497$ reporting LBP in previous 10 years), $n = 2178$	Isometric MVC for torso, arm and leg lift was conducted at baseline 4 year follow-up conducted for claims related to low back injuries or LBP	Participants with higher MVC for arm, leg and torso lift were at higher risk for LBP and low back injury ($p = 0.01, 0.03, \text{ and } 0.26$ respectively). When adjusted for age and sex however no association was present.	Due to an injury rate of 0.6% during torso lift testing it was discontinued. $n = 495$ participants completed torso lift testing, $n = 2158$ completed arm lift testing, and $n = 2102$ completed leg lift testing
Lee et al. (1999)	Healthy student participants without history of LBP, $n = 67$	Isokinetic trunk extension, flexion, and rotation MVC conducted at baseline. 5 year follow-up concerning LBP incidence	27% developed first time LBP during the follow-up period Ratio of extension/flexion strength at baseline was significantly lower in participants who developed first time LBP, ($p < 0.05$)	Age, height, weight and smoking habits similar between groups
Kujala et al. (1996)	Healthy participants without history of LBP, $n = 262$	Standing isometric trunk extension/flexion MVC was conducted at baseline 5 year follow-up with questionnaire was conducted regarding type, frequency, severity and functional limitations of LBP	47% developed first time LBP during the follow-up period, 11% of these reporting it as being of monthly frequency, 17% reporting radiating limb pain, and 2% having been hospitalised due to LBP Trunk extension/flexion was not associated with development of first time LBP	Age, weight and BMI similar between groups Height, occupational physical demands, and occupational musculoskeletal loading was significantly associated with first time LBP ($p < 0.05$)
Chaffin (1978)	Pre-employed plant workers in a	Isometric MVC for torso, arm and leg	As job strength requirements	

	variety of jobs involving manual lifting, $n = 551$	<p>lift in addition to job specific demands was conducted at baseline</p> <p>Preventative effectiveness of strength relative to job demands were evaluated by examining incidence and severity of low back injuries over an 18 month follow-up period</p> <p>Participants were grouped into tertiles relating to their individual strength relative to their job demands</p>	exceeded participant strength the incidence and severity of low back injuries increased at a ratio of 3:1 across the tertiles	
Keyserling (1980)	Pre-employed plant workers applying for a range of 20 varied jobs, $n = 71$	<p>Isometric MVC for torso and arm lift, and push in/out in addition to job specific demands was conducted at baseline</p> <p>Preventative effectiveness of strength relative to job demands evaluated by placing of experimental ($n = 20$) group into jobs matching strength whereas control group ($n = 51$) were not</p> <p>Incidence of musculoskeletal injuries were evaluated over a 1 year follow-up period</p>	During the follow-up period the control group experienced 19 incidences of musculoskeletal injuries compared to 0 in the experimental group	Age, weight and height similar between groups
Salminen et al. (1995)	<p>Healthy children, $n = 38$</p> <p>Children with LBP, $n = 31$</p> <p>Children with LBP and sciatica, $n = 7$</p>	<p>Biering-Sorensen test, sit up isometric test with knees at 90° and MRI conducted at baseline</p> <p>3 year follow-up period evaluating LBP ever, LBP in past 12 months, and recurrent/continuous LBP</p>	Both flexion and extension endurance times were significantly lower in LBP groups ($p < 0.05$) at baseline and follow-up yet endurance time was not predictive of development of first time LBP	Age, sex, school matched between groups
Sjolie & Ljunggren (2001)	Healthy adolescents, $n = 86$	<p>Biering-Sorensen test and questionnaire regarding LBP conducted at baseline</p> <p>3 year follow-up period with questionnaire was completed</p>	High mobility /endurance time ratios were significantly associated with development of LBP at follow-up when adjusted for gender, LBP at baseline, and well-being and physical activity at follow-up (OR 1.5 - 1.9, 95% CI 1.1 – 3.2, $p < 0.05$)	

Adams et al. (1999)	<p>Healthy nurses without history of LBP, $n = 262$</p> <p>Healthy nurses who had previously suffered with 'non-serious' LBP, $n = 141$</p>	<p>Biering-Sorenson test, isometric back lift MVC and back lift at 80%MVC for 20 seconds while EMG recorded from T10 and L3 conducted at baseline</p> <p>3 year follow-up (every 6 months) conducted using questionnaire regarding LBP in previous 6 months</p>	<p>Endurance time at 3 year follow-up was significantly associated with development of serious LBP ($p < 0.01$) and approached significance for any LBP ($p < 0.058$)</p> <p>Neither back lift nor indices of fatigue were associated with development of LBP</p>	
Mostardi et al. (1992)	Healthy nurses without history of LBP, $n = 171$	<p>Isokinetic back lift MVC conducted at baseline</p> <p>Injury reports used to examine incidence of low back injury over 2 years follow-up</p>	<p>9% sustained low back injuries during the follow-up period</p> <p>There was no significant difference in strength at baseline between those who reported low back injury during follow-up and those who did not</p>	
Cady et al. (1979)	Healthy fire-fighters without LBP, $n = 1652$	<p>Isometric back lift MVC conducted at baseline</p> <p>Incidence of prior low back injuries examined subsequent to baseline measurements – no specific follow-up duration was noted</p> <p>Participants were split into percentiles for 'Most Fit' (84-100 percentile), 'Middle Fit' (17-83 percentile) and 'Least Fit' (0-16 percentile)</p>	<p>7.14% sustained low back injuries in the 'Least Fit' group, 3.19% sustained low back injuries in the 'Middle Fit' group, and 0.77% sustained low back injuries in the 'Most Fit' group</p>	Mean age increased with decreasing fitness levels between the three groups
Mooney et al. (1996)	Workers without history of LBP in a ship-building firm in the 3 highest Physical Demand Characteristic categories across 32 jobs, $n = 152$	<p>Isolated lumbar extension MVC using MEDX</p> <p>2 year follow-up of low back injury and LBP claims</p>	<p>9% sustained low back injuries during the follow-up period the majority occurring in the heavy PDC category (64%)</p> <p>Isolated lumbar extension strength was not predictive of low back injuries and only 2 of those participants injured had below normal strength</p>	<p>Age, height and weight was similar amongst PDC categories and in those injured and uninjured</p> <p>Low back injury rates were significantly higher in heavy and very heavy PDC categories ($p < 0.0001$)</p>
Stevenson et al. (2001)	Spinning operators from DuPont without history of LBP, $n = 72$	EMG recorded bilaterally from lumbar paraspinal muscles at T10	EMG indices of fatigues entered final model and were significantly	Other factors in final predictive model included age, peak thoracic

	Spinning operators from DuPont suffering from LBP in previous 2 years, $n = 46$	and L3 level 3-4cm from midline during Biering-Sorenson test	predictive of LBP ($p = 0.035$)	acceleration, leg strength/ endurance, however psychosocial factors were largely absent.
	Spinning operators from DuPont suffering from LBP in previous year, $n = 31$	2 year follow-up period at 6 month intervals for LBP experiences in previous 6 months		
Heydari et al. (2010)	Healthy participants classified as either 'No History of LBP', 'CLBP' or 'Past History of LBP', $n = 105$	EMG recorded bilaterally from lumbar paraspinal muscles at L4/L5 level during back lift test maintaining 2/3MVC for 30 seconds at baseline and follow-up	At follow-up 76 classified themselves as 'the same', 13 'better' and 16 'worse'	
		2 year follow-up participants were asked to classify themselves as 'worse', 'better', or 'the same'	EMG indices of fatigue showed greater fatigue was significantly associated with development of first time LBP and with self-classification at follow-up ($p < 0.05$)	

Maximal Voluntary Contraction = MVC; Cross Sectional Area = CSA; Magnetic Resonance Imaging = MRI; Physical Demand Characteristic - PDC; Low Back Pain = LBP; Body Mass Index = BMI

2.2.7 Discussion

It would appear that there certainly exists evidence indicative of some role specifically of lumbar extensor deconditioning in LBP, which may be causative, yet there is certainly scope for improving earlier studies with more appropriate examination of this relationship. The association of deconditioning specifically of the lumbar extensors in those with chronic LBP, and as a prospective risk factor for development of LBP, has been demonstrated in numerous studies and with various different methods. Studies conducting specific testing of ILEX evidence that weakness appears localised to the lumbar extensor musculature (Cassisi et al., 1993; Holmes et al., 1996; Robinson et al., 1992^b; Nelson et al., 1995; Mooney et al., 1995; Mooney et al., 1997; Boyce et al., 2008) as compared with the quite contrasting evidence utilising TEX. Imaging studies also demonstrate that deconditioning is consistently found in the ES, MF and QL of those with chronic LBP (Hultman et al., 1993; Kamaz et al., 2007; Mengiardi et al., 2006; Danneels et al., 2000). However whether this is level or side specific is unclear (Hides et al., 1994; Barker et al., 2004; Hyun et al., 2007) and it appears that adverse muscle fibre composition is perhaps not present (Crossman et al., 2004). Finally, excessive fatigability of the lumbar extensors in symptomatic participants has been evidenced more objectively through use of EMG fatigue indices analysis (Humphrey et al., 2005; Roy et al., 1989; Mayer et al., 1989^a). Thus it seems that specific deconditioning of the lumbar extensor musculature may be a common factor in LBP lending evidence towards the deconditioning hypothesis and to the speculation of other authors regarding its important role (Smith et al., 2008; Pollock et al., 1989; Carpenter & Nelson, 1999; Jones, 1993; Smith et al., 2011). Further support is shown through prospective studies highlighting that deconditioning may be a risk factor for initial development of LBP (Biering-Sorensen, 1984; Luoto et al., 1995; Mannion et al., 1997^b; Lee et al., 1999; Salminen et al., 1995; Sjolie & Ljunggren, 2001; Adams et al., 1999; Mooney et al., 1996; Stevenson et al., 2001; Heydari et al., 2010).

However, it should be noted that although a body of research exists to support this hypothesis, there also exists some contrasting evidence to refute it which has been

conducted with similarly rigorous methodology (Lariviere et al., 2010^a; Crossman et al., 2004; Gibbons et al., 1997; Da Silva et al., 2005; Adams et al., 1999; Mooney et al., 1996). Indeed we have noted throughout this review the concerns with many of the methodologies employed in much previous research, even amongst the more carefully controlled studies. As such, though the hypothesis is by no means refuted, it still requires further rigorous testing that may be found to either further support or more definitely refute it.

For now however, we contend that the hypothesis presents a convincing explanation of LBP. The evidence reviewed herein is also supported by other areas of research considered as important to determining causality by the criteria put forth by Austin Bradford Hill (1965); criteria such as biological plausibility and experimental reversibility (Van Dieen et al., 2012; Balague et al., 2012). Evidence shows that specifically addressing lumbar extensor deconditioning through ILEX resistance exercise programs in chronic LBP provides significant reductions in pain and disability (Smith et al., 2008; Holmes et al., 1996; Nelson et al., 1995; Mooney et al., 1993; Deutsch, 1996; Park et al., 2000; Lee et al., 2000; Choi et al., 2005; Bruce-Low et al., 2012; Risch et al., 1993; Leggett et al., 1999; Costa, 2010; Carlson & MacKay, 2010; Al-Obaidi et al., 2005; Steele et al., 2013^a). There is also evidence suggesting that improvements in ILEX strength correlate with reductions in pain and disability (Nelson et al., 1995; Steele et al., 2013^a). In addition there is evidence that prospectively addressing lumbar extensor deconditioning through ILEX resistance training reduces risk of further low back injury occurring (Mooney et al., 1995; Matheson & Mooney, 2006; Dreisinger, 2000). Thus there is evidence for a relatively consistent prospective and cross sectional association, biological plausibility through biomechanical modelling studies of lumbar spine stability, experimental reversibility, and also evidence for prospective strengthening to reduce injury risk. These factors combine to offer why deconditioning is perhaps a quite robust account of why LBP is such a wide ranging condition.

One issue that many authors have with this explanation of LBP however is very clearly summarised by Crossman et al. (2004). They note that, of the studies suggesting the presence of lumbar extensor deconditioning in LBP, *“in none of these studies were any mechanisms offered up to explain how “normal” paraspinal muscle could “dysfunction” to predispose to LBP.”* Yet we suggest that the lumbar extensors as an isolated muscle group may exist in a potential state of specific chronic ‘disuse,’ and thus become ‘deconditioned’ in the first instance independent of physical activity levels due to their anatomy (Smith et al., 2008). Indeed this specific state of disuse may stem from the lumbo-pelvic anatomy that is a consequence of our species’ evolutionary history; in essence relatively weak lumbar extensors comparable to strong hip extensors (Lovejoy, 2007). This seems further apparent as most forms of activity and exercise appear to provide little to no conditioning effect (Pollock et al., 1989; Smith et al., 2011; Moffroid et al., 1993; Graves et al., 1994; Mayer et al., 2002^a; Verna et al., 2002; Johnston, 2005; Fisher et al., 2012). Although, as noted in the introduction, ‘disuse’ is often considered as a general reduction in physical activity, it seems here that ‘disuse’ could instead be specifically considered as applicable to the lumbar extensors due to the difficulty in conditioning them, thus leading to their ‘deconditioning’. In a sense, specific ‘disuse’ may lead to specific ‘deconditioning’ of the lumbar extensors, which may further lead to injury and LBP. But this is not simply a reduction in general activity levels; it is due to the inability to effectively maintain their condition as a consequence of their anatomy as the hip extensors appear to ‘take-over’ much of the load bearing (Kankaanpää et al., 1998^a; Clark et al., 2002; 2003).

It should be made clear that it is not the intention of this review to argue for a singular cause of LBP. Although prospective evidence is suggestive of initial deconditioning being a risk factor for development of acute low back injury, LBP and various pain causing mechanisms, and that the majority of acute cases develop into chronic LBP, this is unlikely to be the only potential causative factor. Many other risk factors have indeed been reported. It is even possible that deconditioning is in fact a result of the impact of pain and

other symptoms in some instances (Mannion, 1999) and it is likely that both directions of causality could manifest. That being said, however, a body of evidence would appear to implicate specific lumbar extensor deconditioning in LBP, potentially as a primary factor predisposing injury (figure 3), and thus warrants an addition to the general conceptualisation of the 'Disuse/Deconditioning Syndrome.' This also strongly justifies an exercise based approach designed to effectively recondition the lumbar extensor musculature, regardless of the direction of causality.

That the deconditioning associated with LBP appears for the most part to be mainly localised to the lumbar extensors specifically also warrants that preferably a specific approach towards reconditioning be utilised. Both Helmhout et al. (2008^a) and Mayer et al. (2008) emphasise the issue with many reviews that consider 'exercise' as a single class of treatment without consideration to the variation in exercise approaches that have been applied. Many studies of exercise have also been criticised as lacking an adequate description of the precise exercises used (Helmhout et al., 2008^a; Mayer et al., 2008). Previous Cochrane reviews have not adequately described, defined and categorised the 'exercise' studies they have examined, potentially explaining the generally unfavourable conclusions drawn (Van Tulder et al., 2000; Hayden et al., 2005). The Cochrane reviews have been specifically criticised for this flaw and their wide-sweeping conclusions (Van Tulder et al., 2000; Hayden et al., 2005; Manniche & Jordan, 2001^a; Manniche & Jordan, 2001^b). Indeed we have also raised this issue of specificity (Smith et al., 2008; Steele et al., 2012; Steele et al., 2013^a; Steele et al., 2013^b). As noted, research has shown that the lumbar extensors are notoriously difficult to train unless the pelvis is appropriately restrained in order to provide ILEX (Pollock et al., 1989; Smith et al., 2011; Moffroid et al., 1993; Graves et al., 1994; Mayer et al., 2002^a; Verna et al., 2002; Johnston, 2005; Fisher et al., 2012; Steele et al., 2013^c). That we have presented here that deconditioning may be specifically located in the lumbar extensors supports the contention that exercise approaches should specifically address this.

2.2.8 Conclusion

This review has provided a reconsideration of the importance of the deconditioning hypothesis as it relates to the development of, and association with, LBP. Deconditioning of specifically the lumbar extensors appears to be a consistent factor in LBP. However many of the studies reviewed herein have contained various methodological flaws and so such a conclusion should remain tentative. Future work should seek to further clarify this relationship by acknowledging these and aim to improve upon the previous research. In addition, the results of this review should perhaps be considered in the design of exercise-based rehabilitation approaches to LBP and also as preventative approaches.

2.3 A Review of the Specificity of Exercises Designed for Conditioning the Lumbar Extensors¹³

2.3.1 Overview¹⁴

The objective was to review the specificity of exercises designed to condition the lumbar extensor musculature (i.e. lumbar erector spinae and multifidus). A review of studies examining effects of exercises designed to condition the lumbar extensors was conducted. Included were studies that examined the acute activation and chronic adaptation of the lumbar extensor musculature in response to benches and roman chair trunk extensions, free weights exercises (i.e. deadlifts, squats, good-mornings etc.), floor and stability ball exercise (i.e. trunk extensions, bridging, four-point kneeling etc.) and resistance machines (i.e., those with and without pelvic restraints). Evidence suggests that the reviewed exercises designed to condition the lumbar extensors all may result in significant activation of this musculature during their performance. However, examination of training studies shows that for benches and roman chair trunk extensions, free weights exercises, floor and stability ball exercise and resistance machines without appropriate pelvic restraints, evidence suggests that they may be less effective for inducing chronic adaptations in the lumbar extensors as a result of their performance. Contrastingly, resistance machines that employ appropriate pelvic restraint to isolate lumbar extension are better evidenced to confer specific adaptations to the lumbar extensors. Numerous exercise approaches have been designed with the intention of conditioning the lumbar extensors. Those examined appear to activate the lumbar extensors; however, the specificity of many of these exercises for producing chronic adaptations may be questionable, potentially due to the compound nature of them allowing involvement of other musculature such as the hip extensors. Many of the reviewed exercises offer potential to condition the lumbar extensors, however, isolation of lumbar extension

¹³ Note that this section has been previously published as an independent article by the author; Steele et al., 2013. A Review of the Specificity of Exercises Designed for Conditioning the Lumbar Extensors. *British Journal of Sports Medicine*. Online First

¹⁴ Overview = Abstract amended from the published version.

through appropriate pelvic restraint appears important for optimising specific adaptations in the lumbar extensors.

2.3.2 Introduction

For both athletes and the general population alike, conditioning the muscles of the lower back could be considered an important aspect of physical conditioning. Low back pain (LBP) is a prevalent condition among both the general populace (World Health Organisation, 1998; Office for National Statistics, 2000; 2010; Waddell & Burton, 2000; Walker, 2000; National Institute for Health and Clinical Excellence, 2009) and athletes alike from various sports (Granded & MOrelli, 1988; Sward et al., 1990; Kraft, 2002; Bono, 2004; Bahr et al., 2004). It has been suggested that deconditioning of the lumbar extensor musculature (lumbar erector spinae and multifidus) is a risk factor for low back injury and pain, and that conditioning them through exercise might offer an effective means of reducing this risk (Pollock et al., 1989; Jones, 1993; Carpenter & Nelson, 1999; Smith et al., 2008).

Early efforts at conditioning the lumbar extensors were reported upon by DeLorme and Watkins (DeLorme, 1945; DeLorme & Watkins, 1948). They reported at this time on specialised equipment, designed and intended to provide exercise specifically to the lumbar extensors, by attempting to restrain concurrent pelvic movement. They noted that, with increasing strength, symptoms of LBP were relieved. Studies in occupational settings specifically strengthening the lumbar extensors of employees through preventative exercise have also shown reduced low back injury risk (Mooney et al., 1995; Matheson & Mooney, 2006). Exercise that conditions and strengthens is suggested as valuable in effectively 'prehabilitating' and reducing injury risk for athletes (Stone, 1990). Thus, specifically prehabilitating the lumbar extensors to reduce low back injury and LBP risk might be deemed an important goal of training, and identifying the most effective means an important topic to pursue.

However, the lumbar extensor musculature have been suggested to be notoriously difficult to condition (Pollock et al., 1989; Jones, 1993; Carpenter & Nelson, 1999; Smith et al., 2008). It is further suggested that valid testing of strength or endurance of the lumbar extensors requires isolation of the lumbar spine through restraint of the pelvis (Pollock et al., 1993; Smidt et al., 1983; Petersen et al., 1987; Graves et al., 1990^a; Inanami, 1991; Graves et al., 1992^a; San Juan et al., 2005; Da Silva et al., 2009; Lariviere et al., 2010^a). Otherwise, due to the longer moment arms and relatively larger cross-sectional areas (CSA), the hip extensors (gluteus and hamstrings) may contribute a greater degree of measured torque (Farfan, 1975). This may also influence the effectiveness of exercises designed for the lumbar extensors as during compound exercise the larger musculature is suggested to take over the load (Dul et al., 1984).

Progressive resistance exercise used historically in treating musculoskeletal disorders such as LBP, as well as the suggested difficulty in conditioning the lumbar extensors due to the influence of the hip extensors, has resulted in more specific approaches and devices being developed in order to condition the lumbar extensors. Isolation of the lumbar extensors is potentially important in valid testing (Pollock et al., 1993; Smidt et al., 1983; Petersen et al., 1987; Graves et al., 1990^a; Inanami, 1991; Graves et al., 1992^a; San Juan et al., 2005; Da Silva et al., 2009; Lariviere et al., 2010^a). However, this does not automatically imply exercises that do not isolate are ineffective in conditioning them specifically.

Exercise approaches to conditioning the lumbar extensors have been previously reviewed with regards to treating LBP (Carpenter & Nelson, 1999; Smith et al., 2008; Miltner et al., 2001). However, discussion of the specificity of exercise for conditioning the lumbar extensors was absent from earlier reviews. Mayer et al. (2008) offered the tentative suggestion that isolated lumbar extension (ILEX) exercise using dynamometers appears to be the best option for treating LBP. Though, their conclusion may not be applicable to the consideration of prehabilitation. Thus this present review offers a discussion and

comparison of the specificity of exercise approaches aimed at conditioning the lumbar extensors in asymptomatic populations for prehabilitation purposes. The specificity of exercises in acutely activating the lumbar extensors in addition to their specificity in producing chronic adaptations will be examined.

2.3.3 Methods

Previous reviews' reference lists were searched in addition to SPORTDiscus, PubMed and Google Scholar databases (utilising search terms including combinations of 'lumbar extension' 'lumbar exercise' 'lumbar strength' 'lumbar endurance' 'lumbar spine' 'low back exercise' 'lumbar activation' 'lumbar EMG' as well as other associated terms, synonyms and combinations of the terms) considering all publications listed up to September 2012. Exercise approaches designed to target the lumbar extensors (lumbar erector spinae and multifidus) were defined (see below) and only studies utilising these and providing testing that could isolate and identify the effect of exercise upon the lumbar extensors (also defined below) were included. Thus the results of this review are split into two main sections; 1) effects during performance of exercises upon acute activation, measured using electromyography (EMG), of the lumbar extensor musculature, and 2) training studies reporting chronic adaptation in the lumbar extensors as a result of different exercises. These sections will seek to critically compare the specificity of the exercise approaches examined.

2.3.3.1 Exercises Designed for the Lumbar Extensors

One of the key considerations highlighted by Mayer et al. (2008) is the importance of the actual exercise movement performed to condition the lumbar extensors. Mayer et al. (2008) highlight and define four main types of exercise that are purported to target the lumbar extensor musculature including:

- Benches and Roman Chair Trunk Extensions
- Free Weights (i.e. deadlifts, squats, good mornings etc.)

- Floor and Stability Ball Exercise (i.e. trunk extensions, bridging, four-point kneeling etc.)
- Resistance Machines (i.e those with and without pelvic restraints)

These exercise approaches are defined here as being that they are designed to condition the lumbar extensors. Of these approaches some allow compound trunk extension (TEX) to occur (benches and roman chairs, free weights, floor and stability balls, and some resistance machines) whereas others provide sufficient restraints to isolate lumbar extension (those providing isolated ILEX – described below). Here each of these purportedly ‘specific’ exercises will be considered and both their effect upon acute lumbar extensor recruitment during performance of the exercise, and development of lumbar extensor strength, endurance or hypertrophy through training.

2.3.3.2 Validity of Outcome Measures

Although imaging study perhaps provides the most valid means of identifying whether adaptations have occurred in specific musculature as a result of a training intervention, relatively few studies examining the specificity of lumbar extensor exercises have utilised such outcome measures (Hides et al., 1996; Mooney et al., 1997; Choi et al., 2005). Another means of differentiating the lumbar extensor musculature from, for example, the hip extension musculature, might be to examine changes in fatigability utilising EMG placed over the lumbar extensors during exercise testing. Some studies have utilised such outcome measures (Moffroid et al., 1994). Cross talk is a particular issue when differentiating between the specific lumbar extensor musculature (i.e. lumbar erector spinae and multifidus) and placements (Stokes et al., 2003), however, lumbar electrode placements should be easily distinguishable from hip extensor placements and also from thoracic extensor electrode placements and might be considered to potentially represent the lumbar extensor musculature collectively.

Perhaps explaining the relative paucity of studies using the above mentioned methods, it is often commonplace to determine whether any adaptation has occurred in musculature through use of strength or endurance testing. When examining the lumbar extensors though, consideration should be given to the tests utilised. Testing of compound movements (i.e. TEX) may demonstrate improvement in strength or endurance for a specific movement; but not whether one particular muscle group has improved (Smidt et al., 1983). Isolated testing (i.e. ILEX) instead allows specific induction as to whether the musculature under question has improved (Smidt et al., 1983). Due to the importance of differentiating between such modes of testing for examining the specificity of lumbar extensor exercises they are now explained in advance here.

Because of the potential involvement of the hip extensors in TEX (Lariviere et al., 2010^a) the pelvis should be restrained and stabilised appropriately for the purpose of testing ILEX (DeLorme, 1945; DeLorme & Watkins, 1948; Smidt et al., 1983). If the pelvis is not stabilised then during testing it is difficult to determine the actual source of measured extension torque. Measured torque as a result of TEX may be overstated by the contribution of the hip extensors due to the longer moment arms over which the gluteus and hamstrings exert force and their relatively larger cross-sectional areas (Farfan, 1975). If the pelvis is able to move then measurements of strength obtained may not be valid reflections of the strength of the lumbar extensor musculature.

In addition, the hip extensor musculature has the potential to influence tests of TEX endurance. Measures of endurance using the Biering-Sorenson TEX test show inconsistent results regarding reproducibility (Alaranta et al., 1994; Moffroid et al., 1994; Mayer et al., 1995; McGill et al., 1999; Latimer et al., 1999). Indeed Kankaanpaa et al. (1998^b) reported that the hip extensors show significant fatigue indicative of load sharing during the Biering-Sorensen isometric endurance TEX test. Clark et al. (2002; 2003) have further quantified this suggesting derecruitment of the lumbar extensors and further recruitment of the hip extensors suggesting they are "taking over" the load. Contrastingly,

those that have examined ILEX endurance using appropriate restraint systems for the pelvis (as described below) indicate consistently reproducible results, perhaps due to the control of the specific musculature used during testing (Udermann et al., 2003; Hager et al., 2006).

This involvement of the hip extensor musculature is not just evidenced in prone TEX tests but also in some seated extension test devices (some of which are purported to provide ILEX) where the participant is seated with knees at 90° and the equipment does not restrain the pelvis posteriorly (Kankaanpaa et al., 1998^a). The position of the knees might be important in driving posterior stabilisation of the pelvis against a posterior restraint to specifically isolate the contribution of the lumbar extensors with research suggesting a semi-seated position may be optimal by limiting involvement of both the hip and thoracic extensors (San Juan et al., 2005; Da Dilva et al., 2009; Lariviere et al., 2010^a), and posterior pelvic stabilisation also appearing essential (Smidt et al., 1983; Petersen et al., 1987). Indeed testing with a 90° angle for knee position is shown to overstate torque measures compared with testing that utilises a semi-seated position (Graves et al., 1992^a).

Some manufacturers produce devices that provide the restraint set-up that research suggests is optimal in producing the conditions to test or train ILEX (e.g. Lumbar Extension Machine, MedX, Ocala, Florida; BackUp Dynamometer, Priority One Equipment, Grand Junction, Colorado; Lower Back Revival System, OriGENE Concepts BV, Delft, the Netherlands etc. [Udermann et al., 2003; Hager et al., 2006; Willemink et al., 2012; Smith et al., 2011]). Other researchers have also utilised customised seats providing such optimal restraints to use with generic dynamometers (Da Silva et al., 2009; Lariviere et al., 2010^a; Helmhout et al., 2008^b; Helmhout et al., 2004^a; Harts et al., 2008) suggesting this is also an option available to researchers and clinicians. Force applied by a foot board to the bottom of the feet is transmitted to the distal end of the femurs above the knees through the lower legs. Forces are then transferred in two directions; driving the

femurs towards the rear thus fixing them into the pelvic socket, and force pushing them upwards into knee restraints. A thigh restraint can act as a fulcrum redirecting upwards force at the knees to a downwards force on the pelvic sockets. The results are forces that push the pelvis back and downwards thus preventing any rotation, upward or forward movement (Pollock et al., 1993). If a posterior pelvic restraint which is free to move about its own axis is used then, during consequent flexion and extension, movement of the pelvis is evidenced through rotation of the posterior pelvic restraint (Smith et al., 2008). This restraint set-up ensures isolation of lumbar extension movement from hip extension movement and thus differentiation of the muscles producing each respectively. However, it should be noted in advance that the movement produced during ILEX is a product of both the action of the lumbar extensor musculature (lumbar erector spinae and multifidus) and the thoracic portions of extensor musculature in addition to bilateral contraction of the quadratus lumborum. The relevance of this issue is expanded upon within the discussion.

Isolation of the lumbar extensor musculature through restraining both the legs and pelvis posteriorly has been shown to significantly reduce pelvic movement when compared to less rigorous restraints (Smidt et al., 1983; Petersen et al., 1987) and the restraints described are reported to allow at most only 3° of pelvic rotation (Inanami, 1991), likely as a result of unavoidable soft tissue compliance. As noted by Schmidt et al. (1983), although ultimately the use of both compound and isolated testing is useful in identifying 'weak links,' in the pursuit of an answer regarding the specificity of exercises designed to condition the lumbar extensors a focus should be placed upon isolated testing for chronic outcomes.

2.3.4 Results

2.3.4.1 Acute Activation of the Lumbar Extensors through Specific Exercise

As noted, for valid testing, isolation of lumbar extension requires adequate restraint of the pelvis in order to limit the involvement of the hip extensors (Pollock et al., 1993; Smidt et al., 1983; Petersen et al., 1987; Graves et al., 1990^a; Inanami, 1991; Graves et al., 1992^a;

San Juan et al., 2005; Da Silva et al., 2009; Lariviere et al., 2010^a). However, although this is suggested, it does not provide evidence that the same requirement must be present in order to effectively condition the lumbar extensors. Some exercises do provide such isolation allowing ILEX to be performed, and have been argued to be potentially the most effective means of conditioning the lumbar extensors as a result of a potentially greater activation of the lumbar extensor musculature (Mayer et al., 2008; Smith et al., 2011). Thus it is interesting to examine lumbar extensor activation during other exercises as this may offer insight into the most effective means of training them. Each of the noted types of exercises that are performed with the aim of conditioning the lumbar extensors have been shown to activate these muscles when measured using EMG placements over the lumbar extensors. The results however are quite contrasting both within themselves and in corroboration with data from chronic training studies, as will be discussed.

Roman chair or bench TEX exercise produces significant activation of the lumbar extensors (Kearns et al., 1997; Arokoski et al., 1999; Mayer et al., 1999; Mayer et al., 2002^b; Behm, et al., 2005; Gonclaves et al., 2005; Lariviere et al., 2011), which increases further when internal hip rotation and lumbar extension are accentuated during the movement; this being suggested to better isolate the lumbar extensors involvement (Mayer et al., 2002^b). Indeed, even early analysis of lumbar extensor activation during exercise suggested that hyperextension from the prone position produced greatest activation (Pauly, 1966). Conversely, it has also been shown that when a form of pelvic restraint is used during roman chair TEX, lumbar extensor activation is significantly less than when unrestrained (Benson et al., 2002). This occurred despite participants' subjective perceptions of muscular fatigue being more significantly localised to the lumbar extensors when restraints were used. Arguably the EMG results are more objective yet it is still curious to find this discrepancy. One study has also compared the activation of the lumbar extensors during roman chair TEX to ILEX finding greater activation at lighter loads for the roman chair, and at heavier loads no difference (Kearns et al., 1997). More recently Lariviere et al. (2011) reported greater activation and fatigue of the hip extensors

compared to the lumbar extensors during roman chair TEX, suggesting this was due to greater freedom to change kinematics, which they also reported, and thus load share between the lumbar and hip extensors. Clark et al. (2002; 2003) have supported this apparent load sharing and de-recruitment of the lumbar extensors during TEX demonstrating that, after a certain relative degree of fatigue has been achieved during TEX (~55%), both between further sets of exercise not to failure (Clark et al., 2002) and within a single set of exercise performed to momentary muscular failure (Clark et al., 2003), the lumbar extensors' recruitment decreases. The hip extensors however show evidence of further recruitment and fatigue suggesting they are 'taking over' the load.

Studies have also examined free weight exercises. They have shown exercises such as deadlifts (Escamilla et al., 2002; Hamlyn et al., 2007; Chulvi-Medrano et al., 2010; Colado et al., 2011) and squats (Hamlyn et al., 2007) also produce significant activation of the lumbar extensors with comparative studies suggesting deadlifts to offer the greatest degree of activation (Hamlyn et al., 2007; Colado et al., 2011). Floor and stability ball based exercises (commonly referred to as 'stability' exercises) aimed at conditioning the lumbar extensors also show activation of the targeted musculature, however there is considerable variability between the individual exercises employed using these approaches and even between studies (Arokoski et al., 1999; Behm et al., 2005; Shirado et al., 1995; Vezina & Hubley-Kozey, 2000; Souza et al., 2001; Arokoski et al., 2001; 2004; Mori, 2004; Stevens et al., 2006; 2007). Some degree of activation of the lumbar extensor musculature through these specific exercise approaches appears to occur however and suggests a potential for them to condition them.

The lumbar extensors are also activated when using 'lower back' resistance machines, although there is conflicting evidence as to whether this is greater with or without pelvic restraints and thus TEX or ILEX machines. Udermann et al. (1999) compared lumbar extensor activation during exercise on a machine providing ILEX when the restraints are equipped, both with and without the use of the restraints, finding a trend towards greater

activation in the restrained condition which approached significance ($p = 0.06$). This, however, suggests that the lumbar extensors are, at the least, active in both conditions though perhaps more during ILEX. Walsworth (2004), this time comparing ILEX with another lower back machine that utilised a less sophisticated restraint, reported no significant differences in lumbar extensors activation between the two machines. Two other studies however have suggested the use of an appropriate pelvic restraint system to provide ILEX does result in significantly greater activation of the lumbar extensors (San Juan et al., 2005; Da Silva et al., 2009). San Juan et al. (2005) and Da Silva et al. (2009) reported significantly greater activity in the lumbar extensors when appropriate pelvic restraints are utilised. Both these studies utilised relatively low loads during exercise (50% and 40% of maximal voluntary contraction; MVC, respectively) whereas studies by Udermann et al. (1999) and Walsworth (2004) that have suggested there to be no significant difference in activation of the lumbar extensors utilised greater relative loads (~80% of MVC). Significant lumbar extensor activation observed in an unrestrained yet heavy TEX based movement may be due to the multifidus' role in attempting to stabilise the pelvis due to its origin at the sacrum (Willard, 2007) and this may also explain the findings of Kearns et al. (1997) with regards to Roman Chair extensions showing no difference compared to ILEX at high loads. Another study has shown a resistance machine that fastens only the knees and feet at a 90 degree angle does allow significant activation of the lumbar extensor musculature when using a load of 50%MVC, however, the gluteus maximus may fatigue to a significantly greater degree (Kankaanpaa et al., 1998^a) suggesting that the gluteus maximus may be contributing to the work performed to a greater degree. Contrastingly, Lariviere et al. (2010^a) showed that when a semi seated position and restraints providing ILEX are used there is little activation and fatigue in the hip extensors and that activation and fatigue appears specifically limited to the lumbar extensors.

Despite numerous studies demonstrating the lumbar extensors are active during performance of a variety of different exercises it should be noted this does not provide

direct evidence for adaptations as a result of chronic training; it merely suggests the potential. Indeed some studies demonstrate higher levels of lumbar extensor activation than others depending upon the exercise, the manner in which it is performed, the load and the presence or absence of pelvic restraints. There is also the potential consideration of electrode placements and which of the lumbar extensor musculature (i.e. lumbar erector spinae, multifidus) is being most reflected as it is quite possible that different adaptation could occur in different extensor muscles as a result of different exercises. The involvement of the lower thoracic portion of the extensor musculature is also an important consideration as electrode placements have included those situated on the lower thoracic portion of the extensors (i.e. T9, T10, T11 and T12; Da Silva et al., 2009; Lariviere et al., 2010^a; Arokoski et al., 1999; Lariviere et al., 2011; Escamilla et al., 2002; Chulvi-Medrano et al., 2010), and those ranging across the entirety of the lumbar portion (L1 – S1; San Juan et al., 2005; Da Silva et al., 2009; Kankaanpaa et al., 1998^a; Clark et al., 2002; 2003; Kankaanpaa et al., 1998^b; Kearns et al., 1997; Arokoski et al., 1999; 2001; 2004; Mayer et al., 1999; 2002; Behm et al., 2005; Gonclaves & Barbosa, 2005; Lariviere et al., 2011; Pauly, 1966; Benson et al., 2002; Escamilla et al., 2002; Hamlyn et al., 2007; Chulvi-Medrano et al., 2010; Colado et al., 2011; Shirado et al., 1995; Vezina & Hubley-Kozey, 2000; Souza et al., 2001; Mori, 2004; Stevens et al., 2007; Udermann et al., 1999; Walsworth, 2004) with authors referring to many markers interchangeably as being representative of many of the muscles noted. Gonclaves et al. (2005) reported that there may be differences in fatigue during TEX between electrodes placed over L4/L5 (referred to as the multifidus) and those placed at L2/L3 (referred to as the iliocostalis lumborum) as have others across different placements, both higher and lower, with a range of exercise modes (Da Silva et al., 2009; Lariviere et al., 2011; Hamlyn et al., 2007; Chulvi-Medrano et al., 2010; Udermann et al., 1999). It should again be noted though that there is considerable difficulty in avoiding crosstalk between different electrode locations when examining the lumbar extensors (Stokes et al., 2003) and perhaps this is why other studies have shown that there is little difference between electrode placements in the lumbar region (Kankaanpaa et al., 1998^b; Arokoski et al., 1999; Escamilla et al., 2002;

Vezina et al., 2000; Arokoski et al., 2001; Stevens et al., 2006). Yet in comparing lumbar and lower thoracic extensor activity Arokoski et al. (1999) reported TEX type exercise to produce significant activation in both lumbar and lower thoracic extensors but offered no comparison between placements which appeared similar. However, during fatiguing TEX exercise it would appear that, in tandem with lumbar extensor derecruitment and further hip extensor recruitment (Clark et al., 2002; 2003), there is increased recruitment of the lower thoracic extensors indicating further load modulation between the lumbar and lower thoracic extensors perhaps due to greater freedom of kinematics Lariviere et al., 2011). For free weight deadlift exercise though there appears to be no difference between lumbar or lower thoracic activation (Escamilla et al., 2002; Chulvi-Medrano et al., 2010; Colado et al., 2011). Arokoski et al. (1999) also examined stability exercise but again did not report comparisons between placements. During ILEX exercise it seems there is greater lumbar extensor activity and fatigue compared with the lower thoracic extensors (Da Silva et al., 2009; Lariviere et al., 2010^a). Nevertheless, though comparisons within studies and exercises can be offered in this regard, the range of methodologies used make comparison between studies and exercises difficult.

In addition we must also consider that many trunk muscles demonstrate a potential error in amplitude measurements of ~15% (McGill et al., 1996). Although different treatment of EMG signal data renders difficulty in comparing between studies, the activation levels measured for all of the exercises examined ranged from <5% to ~150% for values normalised to MVC, with between exercise mode comparisons in studies showing differences ranging from ~3% to ~80%. Evidently there are some comparative studies showing clearly significantly greater activation of the lumbar extensors utilising some approaches over others (i.e. greater than 15% difference for bench or ball based TEX compared to other floor/ball based exercises [Arokoski et al., 1999; Behm et al., 2005], deadlift compared to other free weight exercises [Hamlyn et al., 2007; Colado et al., 2011], hip extension or TEX based floor exercises compared to other floor exercises [Souza et al., 2001; Arokoski et al., 2001; Arokoski et al., 2004], and ILEX compared to

machine based TEX [San Juan et al., 2005; Da Silva et al., 2009]), however this is variable between other comparative studies of similar exercises (Benson et al., 2002; Vezina et al., 2000; Stevens et al., 2006; 2007). Thus it is not particularly clear whether all the differences noted can arguably be used to prescribe exercises for conditioning the lumbar extensors. It appears unclear whether some differences in activation reported by studies are indeed present or may be due to measurement errors. Further, the normalised values in studies using TEX based movements for determination of MVC may be of particular concern. During TEX based movement the hip extensors likely contribute significantly more torque during determination of MVC (Pollock et al., 1993; Smidt et al., 1983; Petersen et al., 1987; Graves et al., 1990^a; Inanami, 1991; Graves et al., 1992^a; San Juan et al., 2005; Da Silva et al., 2009; Lariviere et al., 2010^a; Farfan, 1975; Dul et al., 1984), and thus EMG recordings for lumbar extensor MVC could be understated when determined this way i.e. may not be maximal themselves. As such this would affect the relative activation measured in the lumbar extensors during TEX exercise, potentially artificially decreasing the measured activation levels in studies that have used TEX based MVCs (Clark et al., 2002; 2003; Arokoski et al., 1999; Mayer et al., 2002^b; Behm et al., 2005; Benson et al., 2002; Escamill et al., 2002; Hamlyn et al., 2007; Chulvi-Medrano et al., 2010; Colado et al., 2011; Vezina et al., 2000; Souza et al., 2001; Arokoski et al., 2001; Arokoski et al., 2004; Stevens et al., 2006; 2007).

To possibly account for this some studies have utilised analysis of EMG fatigue indices (i.e. EMG power spectrum analysis) which allow examination independent of MVC (Roy et al., 1995). These studies do show that exercises such as TEX and unrestrained seated TEX machines show significant fatigue occurs in the lumbar extensors, but that it also occurs in the hip extensors (Kankaanpaa et al., 1998^a; Clark et al., 2002; 2003; Kankaanpaa et al., 1998^b; Gonclaves et al., 2005; Lariviere et al., 2011) and potentially lower thoracic extensors (Lariviere et al., 2011). Training to momentary muscular failure is supported as an optimal stimulus for muscular conditioning (Rodney et al., 1994; Schott et al., 1995; Drinkwater et al., 2005) as this allows maximal voluntary recruitment of targeted

musculature (~85-100%; Carpinelli, 2008). Despite load sharing of the hip extensors (Clark et al., 2002; 2003) and lower thoracic extensors (Lariviere et al., 2011) and de-recruitment of the lumbar extensors during TEX to failure, in some exercises they may achieve around $\geq 85\%$ of maximal voluntary activation (particularly TEX [Behm et al., 2005], deadlifts and squats [Hamlyn et al., 2007; Chulvi-Medrano et al., 2010; Colado et al., 2011], hip extension or TEX based floor exercises [Arokoski et al., 2001; Arokoski et al., 2004], and both TEX and ILEX based extension machines [San Juan et al., 2005; Walsworth, 2004]) and a significant degree of fatigue (Kankaanpaa et al., 1998^a; Clark et al., 2002; 2003; Kankaanpaa et al., 1998^b; Gonclaves et al., 2005; Lariviere et al., 2011) which suggests the potential for adaptations may indeed exist through these exercises.

Despite potential methodological differences, the contrasting activation provided by different specific exercises may impact upon lumbar extensor conditioning as a result of training using them and thus the value they present in prehabilitation. The degree of activation seen in the lumbar extensors in these studies provides only an indication as to the effectiveness of the exercise for effective strength and endurance adaptations. However, to determine the effectiveness of these exercises in actually conditioning the lumbar extensors over a chronic training period, and thus their value in prehabilitation, it is necessary to examine studies that have utilised a training intervention of exercises and performed testing that allows potential identification of adaptation in the lumbar extensors.

2.3.4.2 Chronic Adaptation to Specific Exercise Training for the Lumbar Extensors

Unfortunately, recommendations regarding effectiveness of a particular exercise upon conditioning the lumbar extensors are often based solely upon whether or not the muscle is active during specific exercise (Hamil & Knutzen, 2007). Yet a number of training intervention studies have in fact examined the effect of exercises aimed at the lumbar extensors upon strength and endurance. However, as noted, to determine whether any adaptation has potentially occurred in the lumbar extensors it is necessary to employ testing of lumbar extension strength or endurance in isolation (ILEX testing) or testing that

allows differentiation between hip extension and lumbar extension (i.e. EMG or imaging study as some have used; Hides et al., 1996; Mooney et al., 1997; Choi et al., 2005; Moffroid et al., 1993; Kamaz et al., 2007).

Nicodemus (1999) highlighted that although some improvement in ILEX strength resulted from roman chair TEX, this was significantly less (~50%) than improvement produced by ILEX exercise. Verna et al. (2002) examined 8 weeks of training using roman chair TEX on the Biering-Sorenson test and ILEX strength. Their results indicated no effect of the training upon ILEX strength. Mayer et al. (2003) reported similar results showing no effect of roman chair exercise upon ILEX. This despite participants in the training group demonstrating an increase in endurance time during the Biering-Sorenson test (Verna et al., 2002). These studies highlight the need for training interventions to confirm effectiveness and specificity of exercises for prehabilitation, in light of contradictory results from acute activation studies (Kearns et al., 1997), (i.e. that acute lumbar extensor activity can sometimes be greater during TEX than ILEX). It seems the considerable degree of freedom to change kinematics (Lariviere et al., 2011) and tendency for the lumbar extensors to de-recruit (Clark et al., 2002; 2003; Lariviere et al., 2011) may explain why studies do not show any adaptation resulting from TEX (Verna et al., 2002; Mayer et al., 2003).

Although not all comparative trials, studies have offered insight into effectiveness of free weight exercise upon the lumbar extensors. Pollock et al. (1989) reported that even healthy strength trained individuals who have engaged in such exercises (deadlifts, squats, good mornings etc.) still demonstrate some degree of lumbar extensor disuse atrophy suggesting that the exercises provide little stimulus to produce or maintain ILEX strength. Pollock et al. (1989) showed considerable increases in ILEX strength after ILEX training in healthy males previously engaged in heavy traditional resistance training for at least 1 year. They speculated that the magnitude of improvement (42-102%; considerably more than reported in studies of other exercises for other muscle groups; ~15-30%) were

due to the lumbar extensors existing in a state of prior atrophy from chronic disuse as most exercises may not stimulate a conditioning effect. However this may also have been due to the highly motivated sample population (Graves et al., 1990^b). A randomised controlled trial has recently been conducted (Fisher et al., 2012) comparing training using ILEX to the stiff-legged deadlift (which loads and significantly activates the lumbar extensors; Escamilla et al., 2002; Colado et al., 2011) upon ILEX strength and deadlift one repetition maximum (1RM). A significant increase in both ILEX strength and deadlift 1RM was observed in the ILEX group after 10 weeks of training whereas the deadlift group only improved deadlift 1RM.

No studies examining the effects of floor and stability exercises upon ILEX strength in asymptomatic populations were found through our literature search. Some have examined effects of floor and stability exercises upon ILEX strength in symptomatic LBP participants (Helmhout et al., 2008^b; Udermann et al., 2004). In addition, studies have examined the effects of such exercise upon multifidus CSA in symptomatic LBP participants (Hides et al., 1996; Danneels et al., 2001). However, it is unclear whether the results of these studies offer any insight as to the use of such exercises for prehabilitation means in asymptomatic participants. A study by Moffroid et al. (1993) has reported the effects of a series of floor based specific exercise, including forms of TEX, on the Biering-Sorenson test and EMG fatigue parameters of the lumbar extensors at the level of L3 in asymptomatic females. They found participants increased endurance times in the Biering-Sorenson test after training but demonstrated no change in fatigability of the lumbar extensors.

Clearly there may be some potential for adaptation to occur in the lumbar extensors through these exercise approaches, though it seems support is equivocal and as such their value in prehabilitation may be questionable or at least requires additional investigation. It appears evidently difficult to stimulate improvement in the lumbar extensors through many supposedly 'specific' exercises. As mentioned, however, some

machines offer means to isolate the lumbar spine providing ILEX and allow the lumbar extensors potentially to be primarily responsible for work performed in isolation (i.e. moving the resistance; Da Silva et al., 2009; Lariviere et al., 2011). However, a number of resistance machines aimed at the lumbar extensors exist commercially. Most of these machines do not employ the rigorous restraint system required for ILEX. However, many have been compared to ILEX in their ability to condition the lumbar extensors.

Results of studies that have compared the effect of commercial 'low back' machines with ILEX machines upon ILEX strength measures are contrasting. Graves et al. (1994) compared training for 12 weeks using an ILEX device to more commercially available lower back machines, both which only restrain the legs. Their results showed, compared with controls, the ILEX group significantly increased ILEX strength, whereas no increase was seen in the other group despite an increased training load reported. Contrastingly, Nicodemus (1999) reported no significant difference in ILEX strength increases when comparing ILEX with another low back extension machine. However, training was only conducted for a period of 5-6 weeks and also the methods including sample size are not reported. Mayer et al. (2002^a) sought to examine whether restraints required for ILEX are indeed required to condition the lumbar extensors. In this study a machine allowing ILEX through equipping of its restraints was used by both training groups to avoid the issue of learning effects confounding the earlier data where the ILEX groups both tested and trained on the same machine (Graves et al., 1994). Thus both groups uses the ILEX machine; one group with the restraints fastened to restrain the pelvis providing ILEX and the other seated in the machine without the restraints fastened properly. Their results suggested the restraint mechanism was not necessary to induce adaptation in the lumbar extensors; however, a concern with their results comes from the small sample size used.

Mayer et al. (2002^a) conducted a power analysis and used both Graves et al. (1994) and Pollock et al. (1989) to determine participant numbers for statistical significance at a power of $\beta = 0.8$. However combining the two studies likely reduced the calculated

participant numbers required due to the effect size (d) observed in Pollock et al. (1989; $d = 4.51$; Calculated using equations from Whitley & Ball [2002]). The large treatment effect of this study (Pollock et al., 1989) has been commented on previously by Graves et al. (1990^b) and noted that the unusually large improvements were due to the asymptomatic participants perhaps being experienced in resistance training and very highly motivated. No other study has demonstrated such large increases. Thus it is likely that the results of Mayer et al. (2002^a) using a relatively small sample size ($n=18$) may represent a type II error (i.e. failure to reject the null hypothesis). On the other hand, Graves et al. (1994) using a larger sample size ($n=77$) was likely more robust and less liable to statistical error. Indeed a recently published study (Smith et al., 2011) that has looked at symptomatic participants employing the same method as the study of Mayer et al. (2002^a) using a greater number of participants ($n=42$) found that the restraint mechanisms used for providing ILEX are necessary to induce adaptation in ILEX strength. Though as with other studies of symptomatic LBP participants it is unclear to what extent the results of this study (Smith et al., 2011) are applicable to the concern of prehabilitation.

Contrastingly however, a study by Parkkola et al. (1992) has demonstrated a significant increase in lumbar extensor CSA at the L4-L5 level through magnetic resonance imaging as a result of TEX based exercise using commercial machines suggesting the possibility of some beneficial effect from such training. It is unclear whether differences in CSA adaptation may occur between approaches however as there is a lack of direct comparison studies.

These studies have examined adaptations in the lumbar extensors as a result of differing exercises. As strength and endurance of the musculature may impact upon injury risk (Mooney et al., 1995; Matheson & Mooney, 2006; Stone, 1990) they provide insight to the potential value of these exercises for prehabilitation of the lumbar extensors in asymptomatic populations.

2.3.5 Discussion

It has been conjectured for some time that when training using compound exercise (i.e. TEX) the larger musculature (i.e. hip extensors; Farfan, 1975), take on greater proportional share of the load and this may affect adaptation produced (Dul et al., 1984; Pauly, 1966). It appears the lumbar extensors are active during exercises such as Roman chair or bench TEXs, free weight exercises such as the squat and deadlift, floor and stability ball exercise, and extension based resistance machines with the pelvis restrained (ILEX) and unrestrained (TEX). However, numerous chronic training studies examining exercises that involve TEX suggest that insufficient stimulus is provided to induce adaptation in the lumbar extensors. Various studies show that roman chair TEXs are significantly less effective compared with ILEX exercise (Nicodemus, 1999; Mayer et al., 2003), though some suggest adaptation might be possible (Danneels et al., 2001; Parkkola et al., 1992). Free weight exercise (i.e. squats, deadlifts) also appear unproductive (Pollock et al., 1989; Fisher et al., 2012). The majority of floor/stability ball based exercise training studies have been conducted in symptomatic participants and contain numerous methodological issues rendering difficulty in drawing conclusion on their effectiveness (Hides et al., 1996; Helmhout et al., 2008^b; Udermann et al., 2004). However, one study in asymptomatic individual's reports that fatigability of the lumbar extensors is not improved using this approach (Moffroid et al., 1993). Finally, studies that have examined low back extension resistance machines appear to offer greater support for an approach that utilises restraint systems for ILEX (Graves et al., 1994; Mayer et al., 2002^a).

There appears to be a distinct discrepancy between studies examining the acute activation of the lumbar extensor musculature and those examining chronic outcomes such as ILEX strength/endurance, change in CSA or change in fatigability. For example approaches such as TEX, free weight exercise such as the deadlift, hip extension and TEX based floor exercises, and unrestrained seated machine extension exercises all demonstrate significant activation of the lumbar extensors which is normally considered as

sufficient for inducing adaptation (Rodney et al., 1994; Schott et al., 1995; Drinkwater et al., 1995; Carpinelli, 2008). The majority of chronic training studies, which have utilised ILEX strength testing in order to examine adaptation in the lumbar extensors, suggest however that these approaches do not effectively condition the lumbar extensors. For example, there is a drastic contrast between the activation levels shown in some studies for the deadlift (~75-125%; Hamlyn et al., 2007; Colado et al., 2011) which does not appear to transfer to improved lumbar extensor strength when tested using an ILEX dynamometer after completion of a 10 week training program utilising deadlifts (Fisher et al., 2012). One possible explanation for this discrepancy shown in many studies might be due to the 'specificity' principle, relating to the exercise used during training and the ILEX testing mode used as an outcome.

In anticipation of this factor affecting measured outcomes, some authors have attempted to examine the purported superiority of ILEX for conditioning the lumbar extensors by utilising the same machine for exercise and testing outcomes both with and without sufficient restraints during acute study (San Juan et al., 2005; Da Silva et al., 2009; Udermann et al., 1999), and also during chronic training studies (Smith et al., 2011; Mayer et al., 2002^a). These studies are contrasting in their findings as noted in the results section of this review with some showing greater activation and strength gains with ILEX (San Juan et al., 2005; Da Silva et al., 2009; Smith et al., 2011), one that does not (Mayer et al., 2002^a), and another that found only a trend towards significantly greater results using ILEX (Udermann et al., 1999). As noted however, those studies suggesting ILEX may not be necessary have methodological limitations, such as smaller sample size, that are of concern. It should also be noted that one of the studies supporting ILEX utilised symptomatic participants (Smith et al., 2011) and so again may not reflect adaptations in asymptomatic participants. Yet, adding further light to the consideration of whether specificity is indeed an issue, an increase in ILEX strength from ILEX training is shown to also produce significant improvement in deadlift 1RM (Fisher et al., 2012). Evidently, improved ILEX strength is associated with improvement in tasks involving TEX; however,

performing these movements may not sufficiently stimulate the lumbar extensors themselves. Reyna et al. (1995) have also shown that maximum ILEX strength correlates well ($r = 0.645$) with dynamic lifting tasks supporting this and highlighting the lumbar extensors may be a weak link (Smidt et al., 1983). Matheson et al. (2002) further reported that ILEX strength independently contributes to lifting capacity. Mooney et al. (1993) also reported significant improvement in lift capacity as a result of 8 weeks ILEX training in chronic LBP participants. That increased lumbar extension strength also improves strength in other compound TEX based movements further reinforces its value in prehabilitation. This is apparently in contrast to what the 'specificity' principle would predict.

Some of the training studies examined utilised either EMG fatigue analysis or measurements of CSA of the lumbar extensors (Moffroid et al., 1993; Parkkola et al., 1992). In particular the study by Moffroid et al. (1993) demonstrated improved Biering-Sorenson test times in the absence of improvements in fatigability of the lumbar extensors. This might suggest that adaptation was limited to the hip extensors potentially to the load sharing they engage in during TEX based exercise (Clark et al., 2002; 2003). However, it is also possible that adaptation may have occurred in the thoracic extensors which have been shown to contribute to load sharing also during TEX exercise (Lariviere et al., 2011). Yet, the majority of chronic training studies have utilised some form of strength or endurance testing and the use of ILEX testing has been emphasised within this review as it allows isolation of lumbar extension movement from hip extension movement. However, it should be noted that the movement produced during ILEX is a product of both the action of the lumbar extensor musculature (lumbar erector spinae and multifidus) and the thoracic portions of extensor musculature in addition to bilateral contraction of the quadratus lumborum. Thus it is noted that although ILEX differentiates clearly between hip extension and lumbar extension, the lumbar extension movement produced is a result of the contribution of the lumbar and thoracic extensors. As such it might be deductively speculated that in those studies involving exercise allowing TEX

movement, and showing improvement in TEX based testing but not in ILEX testing, that adaptations may have occurred primarily in the hip extensors (Verna et al., 2002; Fisher et al., 2012; Graves et al., 1994). An absence of improvement in ILEX would suggest that improvement in neither the lumbar or thoracic extensors has resulted. Contrastingly studies using ILEX exercise interventions do show improved ILEX strength (Graves et al., 1994) though it is more troublesome to discern whether the lumbar or thoracic extensors have improved in these instances. ILEX exercise activation studies do suggest that lumbar extensor involvement is greater than lower thoracic extensor (Da Silva et al., 2009; Lariviere et al., 2011) and therefore that perhaps it is the lumbar extensors adapting to improve ILEX strength. Though in consideration of the discordance between other EMG studies and the chronic training studies examined this appears unsure. 'Thoracic' was not utilised as a term within our search strategy and so it is difficult to draw a fully informed conclusion on the topic of the thoracic extensors adaptations in response to any of the exercises reviewed.

Although evidence is suggestive that ILEX may be a more favourable approach to conditioning the lumbar extensors, future studies should endeavour to more rigorously compare the different exercise approaches noted here in comparison to ILEX exercise. Many have also examined exercise approaches yet not incorporated testing that allows differentiation of the lumbar extensor musculature. These future studies should employ valid, isolated testing of the lumbar extensors either through ILEX strength/endurance testing, EMG fatigue testing of the lumbar extensors, imaging study of adaptations to the lumbar extensors or ideally a combination of these methods. In addition, though there is evidence in occupational settings that prospective strengthening using ILEX reduces injury risk (Mooney et al., 1995; Matheson & Mooney, 2006) and there is reason to believe that conditioning the musculature reduces injury risk (Stone, 1990), it would be of interest for future research to specifically examine this form of 'prehabilitation' in athletic populations.

It is beyond the scope of this review to suggest optimal means of manipulating variables (load, repetitions, repetition duration, volume, frequency) when employing exercises designed for the lumbar extensors. However, it would at present seem there is some evidence to support ILEX exercise to most effectively strengthen the lumbar extensors. Although loaded and active during any of the exercises reviewed, evidently the lumbar extensors and the movement they perform are limited when the pelvis is free to rotate through the action of the hip extensors. Although some have been previously put off by the potentially high cost of some ILEX devices there now exists a range of devices offering such exercise including inexpensive 'low tech' options (e.g. Lumbar Extension Machine, MedX, Ocala, Florida; BackUp Dynamometer, Priority One Equipment, Grand Junction, Colorado; Lower Back Revival System, OriGENE Concepts BV, Delft, the Netherlands etc.). The suggestion to utilise ILEX should not be interpreted as discouraging the use of TEX based exercise as they may provide positive adaptations for the hip extensors (Moffroid et al., 1993; Verna et al., 2002; Fisher et al., 2012; Graves et al., 1994; though again different exercises may offer different degrees of efficacy in this regard; Contreras et al., 2013), and may still have the potential for inducing adaptation of the lumbar extensors as some studies suggest (i.e. TEX using benches or roman chairs [Nicodemus, 1999], hip extension and TEX based floor exercise [Kamaz et al., 2007] and unrestrained machine based TEX [Parkkola et al., 1992; Mayer et al., 2002^b]). However, health and fitness providers and facilities, strength and conditioning coaches, athletes and the general population should consider the specificity of exercises if they have the goal of optimally conditioning the lumbar extensors specifically perhaps as a prehabilitation method for addressing low back injury and pain risk.

2.4 A Review of the Clinical Value of Isolated Lumbar Extension Resistance Training in Chronic Low Back Pain¹⁵

2.4.1 Overview¹⁶

Chronic low back pain (CLBP) is prevalent, costly, and acknowledged as multifactorial in nature. However, deconditioning of the lumbar extensor musculature may be a common factor. Thus specific resistance training is often recommended. Many resistance exercises for the lumbar extensors exist though recent evidence suggests isolated lumbar extension (ILEX) resistance training may best condition these muscles. Thus this review aimed to examine use of ILEX resistance training in participants with CLBP to provide a best evidence synthesis for practitioners and clinicians. Previous reviews' reference lists were searched in addition to SPORTDiscus, PubMed and Google Scholar databases up to May 2014 utilising search terms including combinations and synonyms of 'isolation' 'lumbar extension' 'lumbar exercise' 'lumbar strength' 'lumbar endurance' 'lumbar spine' 'low back exercise' 'CLBP' 'pain' 'disability.' A 'snowballing' style literature search was utilised involving an emergent approach. Studies examining ILEX resistance training as an intervention in symptomatic CLBP populations reporting pain, disability or global perceived outcomes (GPO) as outcomes were examined. Pain and disability were outcomes were compared to consensus guidelines for minimal clinically important changes. Single case reports were excluded. Results suggest ILEX resistance training produces significant and meaningful improvements in perceived pain, disability and GPOs, as part of a multiple intervention or stand-alone approach. A low frequency (1x/week) yet high intensity of effort (to momentary muscular failure) approach using either full or limited range of motion ILEX resistance training appears sufficient and best for significant and meaningful outcomes. Limited comparative studies between ILEX resistance training and other specific exercise approaches exist; however, limited evidence supports ILEX resistance training as more effective. These findings highlight ILEX resistance training as effective for

¹⁵ Note that this section has been previously published as an independent article by the author; Steele et al., 2013. A Review of the Clinical Value of Isolated Lumbar Extension Resistance Training in Chronic Low Back Pain. *PM&R*. In Press

¹⁶ Overview = Abstract amended from the published version.

significant and meaningful improvements in perceived pain, disability and GPOs for CLBP participants. Further research should elucidate comparisons between ILEX resistance training and other specific exercise approaches and clarify whether lumbar extensor conditioning is the mechanism responsible for the improvements reported.

2.4.2 Introduction

Chronic low back pain (CLBP) is one of the most prevalent medical disorders in today's societies (World Health Organisation, 1998; Office for National Statistics, 2000; Waddell & Burton, 2001; Walker, 2000; National Institute for Health and Clinical Excellence, 2009) representing a total economic cost amounting to billions worldwide (National Institute for Health and Clinical Excellence, 2009; Van Tulder et al., 1995; Guo et al., 1999; Maniadakis & Gray, 2000; Waddell et al., 2002; Stewart et al., 2003; Ekman et al., 2005; Ricci et al., 2006; Katz et al., 2006; Freburger et al., 2009). Although CLBP is acknowledged as a multifactorial condition (National Research Council, 1998; National Research Council and Institute of Medicine, 2001) it has been suggested that specific deconditioned extensor muscles of the lumbar spine (lumbar extensor musculature i.e. thoracic and lumbar erector spinae, including the iliocostalis lumborum and longissimus thoracis, the multifidus and also quadratus lumborum when contracted bilaterally) are a risk factor for low back injury and pain (Pollock et al., 1989; Jones, 1993; Carpenter & Nelson, 1999; Smith et al., 2008). Indeed a recent review of the area concluded that persons with CLBP generally present with deconditioning of these muscles identified as reduced lumbar extension strength/endurance, atrophy, and excessive fatigability and that these may be risk factors for low back injury and pain (Steele et al., 2014^a).

Historically, progressive resistance exercise has been recommended for CLBP with the purpose of conditioning the musculature (i.e. developing strength, endurance and hypertrophy; Carpenter & Nelson, 1999; Smith et al., 2008; DeLorme, 1945; DeLorme & Watkins, 1948). The first attempts at providing therapeutic resistance exercise in treating musculoskeletal conditions occurred around the turn of the 20th century (Zander, 1872;

Fischinger et al., 2009). Despite this, mainstream acceptance of progressive resistance exercise was not achieved until around the 1940s by DeLorme and Watkins (DeLorme, 1945; DeLorme & Watkins, 1948). They reported use of specialised equipment used to address the lumbar extensor musculature by attempting to restrict concurrent pelvic movement and found with increasing strength, symptoms of CLBP were relieved (DeLorme & Watkins, 1948). The use of progressive resistance exercise historically in treating musculoskeletal disorders such as CLBP (Carpenter & Nelson, 1999; Smith et al., 2008; DeLorme, 1945; DeLorme & Watkins, 1948), as well as the suggested role of lumbar extensor deconditioning in low back injury and pain (Pollock et al., 1989; Jones, 1993; Carpenter & Nelson, 1999; Smith et al., 2008; Steele et al., 2014^a) has resulted in development of more specific devices for exercising the lumbar extensors. A number of devices exist commercially (e.g. Lumbar Extension Machine, MedX, Ocala, Florida; BackUp Dynamometer, Priority One Equipment, Grand Junction, Colorado; Lower Back Revival System, OriGENE Concepts BV, Delft, the Netherlands), and others have developed customized seats and restraints to use with generic dynamometers (Da Silva et al., 2009; Lariviere et al., 2010^a). All provide isolated lumbar extension (ILEX) through their unique method of restraining the pelvis. The necessary features for achieving ILEX have been described previously (Smith et al., 2008; Steele et al., 2013^c). However, figure 4 presents the restraint system considered as necessary for isolation of lumbar extension. The mechanism of the restraint system should be considered for its ability to specifically isolate and exercise the lumbar extensors. Indeed it has been suggested for some time that specific exercise must be isolated to effectively address the lumbar extensor musculature (Pollock et al., 1989; Jones, 1993; Carpenter & Nelson, 1999; Smith et al., 2008; DeLorme & Watkins, 1948).

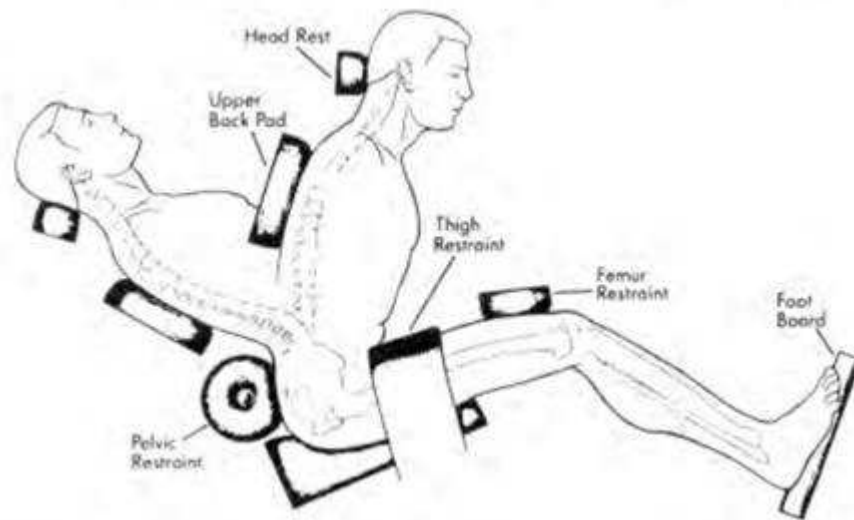


Figure 4. Example of a restraint system used to allow isolated lumbar extension (ILEX) exercise and testing to be performed (Reprinted with permission from MedX Corporation)

However, when exercise is typically examined in relation to CLBP, the varied and different approaches available are often considered in the same category and as being equal (Mayer et al., 2008; Slade & Keating 2006). Specific deconditioning of the lumbar extensor musculature may be an important factor (Steele et al., 2014^a) and thus it is unlikely that all exercise programs are equally effective in addressing CLBP (Mayer et al., 2008; Slade & Keating, 2006; Helmhout et al., 2008^a). Both Helmhout et al. (2008^a) and Mayer et al. (2008) emphasise the issue with many previous reviews examining ‘exercise’ as a single class of treatment without consideration of the variation in exercise approaches that have been used. Many studies of exercise have also been criticised as lacking an adequate description of the precise exercises used (Slade & Keating, 2006; Helmhout et al., 2008^a). Previous Cochrane reviews have not adequately described, defined and categorised the ‘exercise’ studies they have examined, potentially explaining the generally inauspicious conclusions drawn (Van Tulder et al., 2000; Hayden et al., 2005). The Cochrane reviews have been specifically criticised for this flaw and wide-sweeping conclusions (Hayden et al., 2005; Manniche & Jordan, 2001^a; Manniche & Jordan, 2001^b). In a recent meta-regression the authors noted firstly that exercise type may be an important factor that explains the heterogeneity between ‘exercise’ studies, yet due to limitation of the methodology used were unable to analyse the trials included based upon differences in

this characteristic (Ferreira et al., 2010). This issue of specificity of exercise type has also been discussed more recently and continues to be suggested as a potentially important factor to consider (Steele et al., 2012; Steiger et al., 2012^b).

Despite the proposed importance of such specificity in exercise type the necessity of devices to isolate the lumbar extensors for the purposes of specifically conditioning them, and particularly for use in treatment of CLBP, is at present controversial. Many specific exercise approaches for the lumbar extensors have been defined and presented by Mayer et al. (2008). These are considered to be exercises designed to specifically address and condition the lumbar extensors and include; benches and roman chair trunk extensions (TEX), free weights (i.e. deadlifts, squats, good mornings etc.), floor and stability ball exercise (i.e. TEX, bridging, four-point kneeling etc.), and resistance machines including those with and without restraints capable of providing ILEX. However, a recent review has examined the efficacy of these exercises concluding that, though many may offer some degree of lumbar extensor conditioning, ILEX resistance training appears to be most effective for this purpose (Steele et al., 2013^c). Considering the potential role of specific lumbar extensor deconditioning in CLBP (Steele et al., 2014^a) it is of interest to review the efficacy of ILEX in symptomatic populations as it appears to be an approach potentially most effective in addressing this specific factor. Thus the aim was to conduct a mixed review to search and appraise the literature examining the use of ILEX resistance training in participants with CLBP in order to provide a best evidence synthesis for practitioners and clinicians. The intention was to consider 1) studies examining ILEX resistance training's efficacy in this population upon perceived pain, disability and global perceived outcomes (GPO) including the clinical meaningfulness of these outcomes, 2) the manipulation of ILEX resistance training variables for best outcome such as to provide recommendations for clinical prescription, 3) and to examine comparative studies of ILEX

resistance training and other specific exercise approaches¹⁷, including use of ILEX resistance training as part of a multiple or single intervention approach.

2.4.3 Methods

Previous reviews' (Carpenter & Nelson, 1999; Smith et al., 2008; Mayer et al., 2008; Miltner et al., 2001) reference lists were searched in addition to SPORTDiscus, PubMed and Google Scholar databases up to May 2014 utilising search terms including combinations and synonyms of 'isolation' 'lumbar extension' 'lumbar exercise' 'lumbar strength' 'lumbar endurance' 'lumbar spine' 'low back exercise' 'CLBP' 'pain' 'disability.' A 'snowballing' style literature search (Greenhalgh & Peacock, 2005) was utilised involving an emergent approach as the search progressed including searching references of references and utilising personal contact with authors and colleagues knowledgeable in the area. Broadly, any studies examining ILEX resistance training as an intervention in symptomatic CLBP populations reporting pain, disability or GPOs as outcomes were examined. Single case reports were excluded.

2.4.4 Results

Table 5 presents a summary of all the identified studies utilising ILEX that were located and considered in this review.

¹⁷ When referring to specific exercise in this review we are referring to those defined by Mayer et al. (2008). However, a currently standard exercise approach used in addressing CLBP is that of training motor control and the neuromuscular system, which is sometimes referred to as being 'specific' in the sense of training a specific movement. Therefore, to reiterate and clarify for readers of this review in order to avoid confusion, 'specific' exercise in this review refers to exercise approaches designed to specifically target and condition the lumbar extensor musculature and not to motor control based approaches aimed at training the neuromuscular system.

Table 5. Summary of studies examining ILEX in CLBP upon pain, disability and GPOs

Study	Participants	Method	Outcome	Achieved MCICs (Ostelo et al., 2008) for VAS or ODI?	Follow up?
Mooney et al. (1993)	29 females, 26 males with CLBP	<p>All participants underwent an 8 week intervention 2x/week using ILEX resistance training, other resistance training exercises and bike, stair or treadmill exercise.</p> <p>Load, whether exercise was performed to MMF, sets, repetitions, repetition duration, and ROM for ILEX was not reported</p> <p>Pre and post VAS and ODI were completed.</p>	Significant improvement in both VAS (12.3mm – 18.3mm; $p = 0.0001$) and ODI (2.12pts – 2.29pts; $p = 0.001$).	<p>VAS achieved MCIC.</p> <p>ODI failed to achieve MCIC.</p>	N/A
Park et al. (2000)	6 males and 22 females (age ~42 years) with CLBP	<p>Participants underwent an 8 week intervention 2x/week using ILEX resistance training</p> <p>Load was estimated at ~50-70% of max isometric torque and 10 repetitions performed. Whether exercise was performed to MMF, sets, ROM and repetition duration for ILEX was not reported.</p> <p>VAS and daily activity level were completed pre and post.</p>	Significant improvement in VAS (30mm; $p < 0.01$) and daily activity level ($p < 0.05$).	VAS achieved MCIC	N/A
Lee et al. (2000)	29 participants with CLBP	<p>Participants underwent an 8 week intervention 2x/week using ILEX resistance training</p> <p>VAS was completed pre and post.</p>	Significant improvement in VAS (26mm; $p < 0.05$)	VAS achieved MCIC	N/A
Holmes et al. (1996)	18 females (Age 68.2±7.5 years, stature 162.8±7.5 cm, body mass 63.2±10.3 kg) with CLBP	Participants underwent intervention 2x/week for the first 4 weeks reducing to 1x/week if participants did not increase pain during sessions using	Participants significantly improved VAS (~3.2 pts; $p < 0.05$)	VAS achieved MCIC	N/A

		ILEX resistance training		
		<p>A single set of ILEX a load permitting 20 repetitions through their full ROM before MMF using a slow controlled manner taking at least 3-4 seconds for each repetition. Load was progressed once the participant could complete more than 20 repetitions. Load was not reported.</p> <p>VAS (10 pts scale) was completed pre and post</p>		
Steele et al. (2013 ^a)	14 males and 10 females (age ~41-46 years, stature 173-180 cm, body mass 75-85 kg) with CLBP	<p>10 participants underwent a 12 week intervention 1x/week using ILEX resistance training with a full ROM</p> <p>7 participants underwent a 12 week intervention 1x/week using ILEX resistance training with a limited ROM (mid 50% of their full ROM)</p> <p>Both groups performed a single set of ILEX using 80% of their max isometric torque permitting 8-12 repetitions (70-105 seconds) before MMF using a slow controlled manner taking 2 seconds for the concentric phase, holding for 1 second in extension, and 4 seconds for the eccentric phase. Load was progressed by 5% once the participant could complete more than 12 repetitions.</p> <p>7 participants acted as non-training controls</p> <p>VAS and ODI were completed pre and post</p>	Both ILEX groups significantly improved in VAS (~16-30mm) and ODI (~12-18pts) compared to the control group ($p < 0.05$) with no significant difference between ILEX groups.	VAS and ODI achieved MCIC N/A

Choi et al. (2005)	38 males and 37 females (age ~42-51 years, stature ~165 cm, body mass ~63-67 kg) undergoing first time lumbar discectomy for disc herniation not responding to conservative treatment	<p>35 participants underwent a 12 week intervention 6 weeks post-surgery using ILEX resistance training, other resistance training exercises and aerobic exercise.</p> <p>Load, whether exercise was performed to MMF, sets, repetitions, repetition duration, and ROM for ILEX was not reported</p> <p>40 participants constituted a control group completing 12 weeks of home-based lumbar conditioning exercises</p> <p>No details of home-based exercises were reported</p> <p>VAS and ODI were completed pre and post and during follow-up. Return to work 4 months after surgery was also reported.</p>	<p>ILEX group improved significantly more compared to the control group in VAS at the end of the 12 week intervention (ILEX group 57mm, Control group 38mm).</p> <p>No significant difference between groups for change in ODI</p>	VAS and ODI achieved MCIC	<p>At 4 months post-surgery 87% of the ILEX group had returned to work compared to 24% of the controls</p> <p>At 6 months post-surgery ~92% of both groups had returned to work</p> <p>At 1 year follow up VAS was similar between groups</p>
Smith et al. (2011)	42 participants (age 42.93±10.80 years) with CLBP	<p>15 participants underwent a 12 week intervention 1x/week using an ILEX resistance training with the restraints fastened (STAB).</p> <p>15 participants underwent a 12 week intervention 1x/week using an ILEX resistance training without the restraints fastened (NO-STAB).</p> <p>Both groups performed a single set of ILEX using a load that permitted 8-12 repetitions before MMF through a full ROM using a slow controlled manner taking 2 seconds for the concentric phase and 4 seconds for the eccentric phase. Load was progressed</p>	<p>STAB significantly improvement in both VAS (~17mm; $p < 0.01$) and ODI (~12pts; $p < 0.01$). No change was observed for NO-STAB or control groups for either VAS or ODI.</p>	VAS and ODI achieved MCICs in the STAB group	N/A

		by 5% once the participant could complete more than 12 repetitions.		
		12 participants acted as non-training controls		
		VAS and ODI were completed pre and post		
Ju et al. (2012)	14 participants (age ~45 years, stature ~162 cm, body mass ~63 kg) undergoing lumbar disc herniation surgery	<p>7 participants underwent a 12 week intervention 3x/week post-surgery using ILEX resistance training and other resistance training exercises</p> <p>ILEX was performed using 40-50% of max isometric torque for 18-20 repetitions. Load was progressed based upon results of retesting every 4 weeks. Sets, repetition duration, and ROM for ILEX was not reported.</p> <p>7 participants constituted a control group completing rest and utilising conservative treatments.</p> <p>VAS for back pain. Night pain, exercise pain and handicap were completed pre and post.</p>	<p>ILEX group improved significantly in all VAS measures at the end of the 12 week intervention (back pain ~7.6 mm, night pain 9.3 mm, exercise pain 27.5 mm, handicap 29.9 mm; all $p < 0.05$).</p> <p>The control group made no significant improvement.</p>	VAS for back pain did not meet MCIC N/A
Bruce-Low et al. (2012)	42 males and 30 females (age 45.5 ± 14.1 years) with CLBP	<p>31 participants underwent a 12 week intervention 1x/week using ILEX resistance training</p> <p>20 participants underwent a 12 week intervention 2x/week using ILEX resistance training</p> <p>The 1x/week group performed a single set of ILEX using 80% of their max isometric torque permitting 8-12 repetitions (70-</p>	<p>Both ILEX groups significantly improved in VAS (~16-21mm) and ODI (~12-15pts) compared to the control group ($p < 0.05$) with no significant difference between ILEX groups.</p>	VAS and ODI achieved MCIC N/A

		<p>105 seconds) before MMF through a full ROM using a slow controlled manner taking 2 seconds for the concentric phase, holding for 1 second in extension and 4 seconds for the eccentric phase. Load was progressed by 5% once the participant could complete more than 12 repetitions.</p> <p>The 2x/week group performed the same session as above in addition to performing a single set of ILEX using 50% of their max isometric torque permitting 15-20 repetitions (105-140 seconds) before MMF through a full ROM using a slow controlled manner taking 2 seconds for the concentric phase, holding for 1 second in extension and 4 seconds for the eccentric phase. Load was progressed by 5% once the participant could complete more than 20 repetitions.</p> <p>21 participants acted as non-training controls</p> <p>VAS and ODI were completed pre and post</p>			
Stephan et al. (2011)	74 participants (55% females, age ~44 years) with CLBP	<p>58 Participants underwent an intervention lasting and average ~24.5 weeks of average ~1.6x/week using ILEX resistance training and other resistance training exercises.</p> <p>ILEX and other exercises were performed for a single set using 60% of their 1 repetition maximum permitting 6-9</p>	<p>Significant reductions in VAS, pain severity, effects of pain and ODI were seen at 3 and 6 months (all $p < 0.001$).</p> <p>The control group significant reduced ODI at 3 months ($p < 0.05$) and pain severity at 6 months ($p < 0.05$) but did not significantly change in any other measure.</p>	Both VAS and ODI met MCIC	N/A

		<p>repetitions stopping prior to MMF for sessions 1-20 and achieving MMF from session 21 onwards, using a slow controlled manner taking 4 seconds for the concentric phase, holding for 2 second in extension, and 4 seconds for the eccentric phase through full pain free ROM. Load was progressed was not reported.</p> <p>18 participants acted as non-training waiting list controls</p> <p>VAS, pain severity and effects of pain were measured using the MOS in addition to ODI were completed at 3 and 6 months.</p>			
Kim et al., (2010)	40 male patients undergoing surgery for lumbar discectomy (Age ~40 years, stature ~173 cm, body mass ~75kg)	<p>All patients underwent lumbar discectomy followed by 6 weeks of rest.</p> <p>After lumbar discectomy and 6 week rest all participants underwent a 12 week intervention 2x/week using ILEX resistance training</p> <p>After completion of the initial 12 week intervention:</p> <p>10 participants underwent a 12 week intervention 2x/week using ILEX resistance training (Group 1)</p> <p>10 participants underwent a 12 week intervention 1x/week using ILEX resistance training (Group 2)</p> <p>10 participants underwent a 12 week intervention 1x/2weeks using ILEX resistance training</p>	<p>Group 3 did not improve in either VAS or ODI</p> <p>Group 1 and 2 both significantly improved in ODI (0.8 to 1.4 pts; $p < 0.05$)</p> <p>Only Group 1 significantly improved VAS (0.5 cm; $p < 0.05$)</p>	VAS and ODI did not meet MCICs	N/A

		(Group 3)			
		Each group performed 2 sets of ILEX permitting 15-20 repetitions taking 3 seconds for the concentric phase, and 3 seconds for the eccentric phase. Load, ROM and progression for ILEX was not reported			
		10 participants acted as non-training controls			
		VAS (for LBP and leg pain) and ODI were completed post-surgery and initial 12 week intervention and again post the further 12 week intervention			
Risch et al. (1993)	34 males and 20 females (age ~45 years range 22-70) with CLBP	31 participants underwent a 10 week intervention using ILEX resistance training 2/week for the first 4 weeks, 1x/week for the last 6 weeks. A single set of ILEX was performed using 50% of their max isometric torque performed to MMF through a full ROM. Load was progressed by 5 ft.lb once the participant could complete more than 12 repetitions. Repetition duration for ILEX was not reported 23 participants acted as a waiting list control group. Both completed pre and post West Haven Yale Multidimensional Pain Inventory.	In the intervention group there was a significant improvement in pain subscale of West Haven Yale Multidimensional Pain Inventory (~0.5; $p < 0.002$). No significant changes occurred for the control group.	N/A	N/A
Nelson et al. (1995)	484 males (mean age 38.7 years) and 411 females (mean age 37.1 years) with CLBP were initially	627 participants completed an average of 18 sessions 2x/week using ILEX resistance	In the intervention group 64% and 62% reported substantial decrease in pain, 14% and 17%	N/A	At 1 year follow up 94% of participants with good or excellent results

	recruited	<p>training, other resistance training exercises and aerobic exercise.</p> <p>ILEX was performed alternating between sessions to MMF and sessions not to MMF.</p> <p>Load, sets, repetitions, repetition duration, and ROM for ILEX was not reported</p> <p>107 participants acted as non-training controls.</p> <p>All participants underwent educational sessions and were given a home-based exercise program to utilise during follow-up</p> <p>Pre and post pain was measured using a 5 item scale as well as GPOs. Return to work initially and at 1 year follow-up was also reported.</p>	<p>reported a decrease in pain, 6% and 6% reported a slight decrease in pain, 12% and 13% reported no change in pain, and only 3% and 2% reported a worsening of their pain.</p> <p>The intervention group reported GPO's of 46%, 30%, 14% and 8% for 'excellent,' 'good,' 'fair' or 'poor' respectively.</p> <p>Of 139 participants off work due to CLBP (~73 days) 72% returned to work at completion of the ILEX intervention</p>	<p>maintained improvement, 6% did not change or worsened. Of participants with fair or poor results 25% improved, 75% did not change or worsened</p> <p>Return to work at 1 year follow-up was at 77%</p>
Leggett et al. (1999)	192 males (age ~39-49 years) and 220 females (age ~39-51 years) with CLBP	<p>Participants underwent an 8 week intervention 2x/week using ILEX resistance training, other resistance training exercises, aerobic exercise and McKenzie therapy.</p> <p>ILEX was performed using 50% of their max isometric torque performed to MMF through a full ROM. Load was progressed by 2-5% once the participant could complete more than 15 repetitions. Sets and repetition duration for ILEX was not reported</p> <p>SF36 and GPOs were completed pre and post.</p>	<p>Significant improvement in all subscales of SF36 ($p < 0.0001$). ~74% to ~82%, ~12% to ~24% and ~1% to 5% rated their outcome as either 'better,' 'same' or 'worse' between the two centres used</p>	<p>N/A</p> <p>At 1 year follow up maintenance of outcomes was apparent</p>

Costa (2010)	4 males and 5 females (age ~63 years) with CLBP	<p>Participants underwent an 8 week intervention 2x/week using ILEX resistance training and other resistance training exercises.</p> <p>A single set of ILEX was performed for 8-12. Load was progressed based upon participant's perception as exercise became easier. Load, repetition duration, and ROM for ILEX was not reported</p> <p>McGill Pain Questionnaire and ODI were completed pre and post.</p>	Significant improvement in ODI (5.33pts; $p = 0.033$) but not in McGill Pain Questionnaire (3.22pts; $p = 0.159$)	ODI failed to achieve MCIC.	N/A
Carlson & MacKay, (2010)	28 males (age ~47 range 25-80 years) and 27 females (age ~46.9 range 26-73 years) with CLBP	<p>Participants underwent a 6 week intervention 2x/week using ILEX resistance training.</p> <p>ILEX was performed using a load that permitted 6-9 repetitions before MMF through a full ROM using a slow controlled manner taking 4 seconds for the concentric phase, holding for 2 seconds in extension and 4 seconds for the eccentric phase. Load was progressed by 5% once the participant could complete more than 12 repetitions.</p> <p>Load, sets, and ROM for ILEX was not reported</p> <p>ODI was completed pre and post.</p>	Significant improvement in ODI (9-10.8pts; $p < 0.05$)	ODI achieved MCIC	N/A
Al-Obaidi et al. (2005)	42 participants were initially recruited, 22 males (age 45 ± 6.2 years) and 20 females (age 39.25 ± 5.8 years) with CLBP	<p>36 participants underwent a 10 week intervention 1x/week using ILEX resistance training.</p> <p>A single set of ILEX was performed using a load</p>	RMDQ scores significantly improved (~4 pts, ~16%; $p < 0.001$).	<p>RMDQ achieved MCIC</p> <p>Participants were however dichotomised individually as to whether MCIC was met and fear avoidance</p>	N/A

		<p>permitting 6-12 repetitions before MMF using a slow controlled manner throughout the full ROM. Load was progressed by 5% once the participant could complete more than 12 repetitions. Load and repetition duration was not reported.</p> <p>RMDQ was completed pre and post</p>		beliefs and baseline pain shown to be higher in those failing to achieve MCIC	
Willeminck et al. (2012)	20 participants (Age 46.2±9.7 years) with CLBP	<p>Participants underwent an ILEX resistance training intervention lasting ~24 weeks including 10 session during the first 12 weeks and sessions at participants convenience for the second 12 weeks</p> <p>4 sets of ILEX for 10 repetitions were performed at a load determined by the physiotherapist through a full ROM using a slow controlled manner taking 2 seconds for the concentric phase, and 3 seconds for the eccentric phase. Load was progressed once the participant could complete 4 sets comfortably</p> <p>RMDQ, GPO and patient functional scale (PFS) were completed pre, at 12 weeks post and 24 weeks post.</p>	<p>RMDQ significantly improved at both week 12 and 24 (~3 pts, ~13%; $p = 0.024$)</p> <p>PFS significantly improved at both week 12 and 24 (~70 pts; $p < 0.001$)</p> <p>GPO showed complete recovery or significant improvement in 43.8% and 50.0% at weeks 12 and 24 respectively</p>	RMDQ achieved MCIC	N/A
Helmhout et al. (2004 ^a)	81 male working army participants (age ~40 years) with CLBP	<p>Participants underwent a 10 week intervention 2x/week for weeks 1-2 and 1x/week for weeks 3-12 using ILEX resistance training either as 'High Intensity' (HIT) or 'Low Intensity' (LIT).</p> <p>For HIT load was 35% of max</p>	<p>No significant differences between groups were found for self-assessed improvement, RMDQ, ODI or SF36.</p> <p>TSK was significantly greater in LIT midway through the intervention (~0.4pts; $p = 0.03$).</p>	ODI achieved MCIC for both groups	<p>No significant differences between groups were found for self-assessed improvement, RMDQ, ODI or SF36 at 6 or 9 months follow-up.</p> <p>TSK was significantly</p>

		<p>isometric torque and 15-20 repetitions performed during weeks 1-2 and 10-15 repetitions performance weeks 3-12. Load was progressed by 2.5kg once the participant could complete more than 20 repetitions. Whether exercise was performed to MMF, sets, repetition duration and ROM for ILEX was not reported.</p> <p>For LIT load was 20% of max isometric torque and 15 or 20 repetitions performed during weeks 1-2 and weeks 3-4 after each test. Whether exercise was performed to MMF, sets, repetition duration and ROM for ILEX was not reported.</p> <p>RMDQ, ODI, TSK and SF36 were completed pre and post. Follow-up was conducted at 6 and 9 months.</p>	<p>Lumbar extension strength was significantly greater for HIT at all time points (~31-58 Nm; $p < 0.001$).</p>	<p>greater in LIT at 9 months follow-up (~3.4pts; $p = 0.03$).</p> <p>Lumbar extension strength was significantly greater for HIT at 6 and 9 months follow-up (~24-29 Nm; $p < 0.05$).</p>
Helmhout et al. (2008 ^b)	<p>107 male working army participants (age ~35-37 years, stature ~183 cm, body mass ~85kg) with sub-acute LBP or CLBP</p>	<p>61 participants underwent a 10 week intervention 2x/week using ILEX resistance training.</p> <p>Load was estimated at ~50-70% of max isometric torque and 15-20 repetitions performed in a slow controlled manner taking 2 seconds for the concentric phase and 4 seconds for the eccentric phase. Load was progressed by 2.5kg once the participant could complete more than 20 repetitions. Whether exercise was performed to MMF, sets, and ROM for ILEX was not reported</p> <p>46 participants underwent a 10 week intervention using</p>	<p>No significant between groups differences for improvements in RMDQ (~4-5pts), PSFS (~60mm) at any time.</p>	<p>RMDQ achieved MCIC for both groups</p> <p>Follow up conducted at 36 and 62 weeks showed that improvements were maintained for both groups with no between group differences</p>

		<p>Regular Physiotherapy.</p> <p>Regular Physiotherapy included including 65% of activities as exercise (i.e. trunk and leg strengthening - though physiotherapists were instructed to not use the specific lumbar extension device, core stability exercises, stretching and specific McKenzie exercise), 25% constituted aerobic activity, 10% instruction and advice, and less than 1% as passive modalities.</p> <p>RMDQ, and Patient Specific Functional Score (PSFS), were completed pre and post and during follow-up</p>			
Harts et al. (2008)	65 male working army participants (age ~42 years) with CLBP	<p>Participants underwent a 8 week intervention 2x/week for weeks 1-2 and 1x/week for weeks 3-8 using ILEX resistance training either as 'High Intensity' (HIT) or 'Low Intensity' (LIT) or a waiting list control (WLC).</p> <p>For HIT load was 50% of max isometric torque and 15-20 repetitions performed. Load was progressed by 2.5kg once the participant could complete more than 20 repetitions. Whether exercise was performed to MMF, sets, repetition duration and ROM for ILEX was not reported.</p> <p>For LIT load was 20% of max isometric torque and 15 or 20 repetitions performed. Whether exercise was performed to MMF, sets, repetition duration</p>	<p>HIT group significantly improved in SF36 compared to both LIT and WLC (7%; $p < 0.05$).</p> <p>HIT group significantly improved in self-assessed decrease also compared to both LIT (39%; $p < 0.05$).</p> <p>No significant differences were found for any other variables.</p>	RMDQ did not meet MCIC	<p>No significant differences between groups were found for self-assessed improvement, RMDQ, or SF36 at 16 weeks follow-up.</p>

		and ROM for ILEX was not reported.			
		RMDQ, TSK and SF36 were completed pre and post. Follow-up was conducted at 6 and 9 months.			
Udermann et al. (2004)	9 females (age 39.1±2.8 years, stature 164.2±1.6 cm, body mass 69.3±4.0 kg), 9 males (age 45.0±2.5 years, stature 180.6±1.6 cm, body mass 87.8±4.7 kg), with CLBP	<p>9 participants underwent a 4 week intervention 1x/week using ILEX resistance training.</p> <p>Load was 50% of max isometric torque and a single set of 18-20 repetitions was performed to MMF through a full ROM using a slow controlled manner taking 2 seconds for the concentric phase and 4 seconds for the eccentric phase. Load was progressed by 5% once the participant could complete more than 20 repetitions.</p> <p>9 participants underwent a 4 x/week intervention 2x/week using McKenzie therapy and home exercises every 2 hours.</p> <p>SF36 was completed pre and post.</p>	Significant improvement in 6 of 8 subscales of the SF36 for both groups ($p < 0.05$) with no difference between groups.	N/A	N/A
Vincent et al. (2014)	49 obese participants (67% females, age ~68 years) with CLBP	<p>18 Participants underwent a 4 month intervention 3x/week using ILEX resistance training.</p> <p>17 Participants underwent a 4 month intervention 3x/week using ILEX resistance training and other resistance training exercises.</p> <p>ILEX and other exercises were performed for a single set using 60% of their 1 repetition for 15 repetitions attempting to</p>	Significant group by time interactions for ODI ($p = 0.015$), RMDQ ($p = 0.007$) and PCS ($p = 0.002$) in favour of the full body group.	Full body met ODI MCIC	Pairwise comparisons between groups were not reported.

produce a rating on the Borg scale of 16-18 Load was progressed 2% per week to maintain this. Whether exercise was performed to MMF, and ROM was not reported

14 participants acted as non-training waiting list controls who underwent standard care (including bodyweight resistance exercises, dietary information and information about back pain).

ODI, RMDQ, Pain Catastrophising, TSK, and fear avoidance beliefs were completed at pre and post.

Isolated Lumbar Extension = ILEX; Range of Motion = ROM; Momentary Muscular Failure = MMF; Chronic Low Back Pain = CLBP; Low Back Pain = LBP; Visual Analogue Pain Scale = VAS; Oswestry Disability Questionnaire = ODI; Roland Morris Disability Questionnaire = RMDQ; Tampa Scale for Kinesiophobia = TSK; Short Form 36 health questionnaire = SF36; Global Perceived Outcome = GPO; Medical Outcomes Study = MOS; Minimal Clinically Important Change = MCIC

2.4.4.1 Pain, Disability and Clinical Meaningfulness of Outcomes

The most common measurement of pain is the visual analogue scale (VAS; Ogon et al., 1996). Several studies have examined the use of ILEX resistance training upon perceptions of pain through this measurement. Many have been designed as prospective single arm trials of symptomatic participants with intervention periods of 8 to 12 weeks and training frequencies of 1 to 2x/week (Mooney et al., 1993; Park et al., 2000; Lee et al., 2000; Holmes et al., 1996). Samples sizes ranged from 18 to 55 participants indicating sufficient power to detect significant changes in VAS (Steele et al., 2013^a) with all reporting significant reductions (Mooney et al., 1993; Park et al., 2000; Lee et al., 2000; Holmes et al., 1996). Other studies have adopted randomised controlled trial designs utilising a non-training control group comparison to confirm the treatment effect from including ILEX resistance training as an intervention (Steele et al., 2013^a; Choi et al., 2005; Smith et al., 2011; Ju et al., 2012; Bruce-Low et al., 2012; Stephan et al., 2011). These studies used interventions of ~12 to 24 weeks with varying frequencies of 1 to 2x/week and sample sizes ranging from 14 to 74 participants again suggesting sufficient power. All reported that, compared with the non-training control groups, the groups performing ILEX resistance training made significant reductions in VAS (Steele et al., 2013^a; Choi et al., 2005; Smith et al., 2011; Ju et al., 2012; Bruce-Low et al., 2012; Stephan et al., 2011). Control groups in these studies were either instructed to perform home based exercise (Choi et al., 2005) continue with any conservative treatments they were already undergoing (Steele et al., 2013^a; Smith et al., 2011; Ju et al., 2012; Bruce-Low et al., 2012) or acted as waiting list controls (Stephan et al., 2011). A study by Kim et al. (2010) examined the effects of varying frequency of ILEX resistance training over 12 weeks upon 40 participants undergoing lumbar discectomy. They reported significant improvement in VAS for ILEX resistance training when training 1 or 2x/week.

Other methods of measurement have also been used to examine the effects of ILEX resistance training upon pain. In a randomised controlled trial of 54 participants Risch et

al. (1993) showed significant improvement as a result of 10 weeks of ILEX resistance training in the pain subscale on the West Haven Yale Multidimensional Pain Inventory when compared to a waiting-list control group. In a large single arm trial involving outcomes from 677 participants who underwent ~9 weeks of ILEX resistance training 2x/week, Nelson et al. (1995) reported participant low back pain and leg pain outcomes using a 5 item scale ('worse,' 'no change,' 'slight decrease,' 'decreased,' 'substantial decrease'). For low back pain and leg pain respectively, 64% and 62% reported substantial decrease, 14% and 17% reported a decrease, 6% and 6% reported a slight decrease, 12% and 13% reported no change, and only 3% and 2% reported a worsening of their symptoms. There was a moderate but significant correlation between the improvements in lumbar extension strength and low back pain ($r = -0.318$) and this relationship appeared even more pronounced when participants were grouped based upon the above categories. Steele et al. (2013^a) also reported significant relationships between improvements in lumbar extension strength and low back pain (VAS) as a result of ILEX resistance training ($r = -0.488$ to -0.668). Another single arm trial conducted by Leggett et al. (1999) across two independent treatment centres showed significant improvements in the pain subscale of the Short Form 36 health questionnaire (SF36; the SF36 is a common outcome that covers a wide range of possible subscales thus presenting an overall 'global picture' of participant well-being). Costa (2010) in a small study involving 9 participants used the McGill Pain Questionnaire and reported a non-significant improvement (-3.22 , $p = 0.159$) which would appear, in light of other research showing significant improvements in pain, perhaps a result of low study power. Stephan et al. (2011) examined the effects of ILEX resistance training upon pain severity and effects of pain using the Medical Outcome Scale reporting significant improvements at both 3 and 6 months stage of the intervention compared with a waiting list control.

Measures of perceived disability, such as the Oswestry Disability Index (ODI; Fairbank et al., 1980) amongst others have also been measured in response to ILEX resistance

training interventions. Mooney et al. (1993) showed a significant improvement in ODI score between pre and post measures for 55 participants undergoing 8 weeks of ILEX resistance training 2x/week. Other single arm trials have also reported significant improvements in ODI including Costa (2010; in contrast the lack of significant results for the McGill Pain Questionnaire), and Carlson & Mackay (2010) over a 6 week intervention of ILEX resistance training 2x/week for 55 participants. Randomised controlled trials again have examined this effect on ODI scores as a result of the intervention in comparison to non-training controls for ~12 to 24 week interventions of ILEX resistance training 1 and 2x/week with samples ranging 24 to 74 participants (Steele et al., 2013^a; Smith et al., 2011; Ju et al., 2012; Bruce-Low et al., 2012; Stephan et al., 2011). Again these studies are sufficiently powered to detect changes in ODI (Steele et al., 2013^a) with all showing significant reductions. It was also reported that significant relationships exist between improvements in lumbar extension strength and disability ($r = -0.414$ to -0.539 ; Steele et al., 2013^a). Choi et al. (2005) noted a non-significant improvement in ODI score that favoured the use of ILEX resistance training compared with non-training controls in post-surgery lumbar discectomy participants; however p values were not reported. Kim et al. (2010) also demonstrated significant improvement in ODI from 12 weeks of ILEX resistance training 2x/week for participants undergoing lumbar discectomy.

Other measures of self-reported disability demonstrate similar results. In single arm trials Al-Obaidi et al. (2005) showed significant improvement in overall group mean between pre and post measures using the Roland Morris Disability Questionnaire (RMDQ) for 42 participants undergoing 10 weeks of ILEX resistance training 1x/week, as did Willemink et al. (2012) for 20 participants undergoing ~24 weeks of ILEX resistance training at a variable frequency. Willemink et al. (2012) however also examined change in multifidus cross sectional area reporting no change. Randomised controlled trials have also examined the RMDQ. Helmhout et al., (2004^a; 2008b) Harts et al. (2008) reported significant improvements in RMDQ in trials of 65 to 107 participants examining 8 to 10

weeks of ILEX resistance training 1 to 2x/week. These studies also compared both heavy and light load ILEX resistance training, waiting list controls and regular physiotherapy which are detailed further below. Risch et al. (1993) also examined the perceived psychological and psychosocial effects of strengthening using ILEX resistance training compared with a non-training control group. Both subscales of the Sickness Impact Profile (Physical and Psychosocial Dysfunction) showed significant improvement as result of the ILEX resistance training intervention. These improvements in perceived dysfunction occurred without any change in psychological variables such as anxiety and stress. Park et al. (2000) also reported a spontaneous increase in daily activity levels as a result of 8 weeks of ILEX resistance training 2x/week which suggested reduced disability or greater willingness to be active.

In terms of GPOs differing approaches have been reported. Nelson et al. (1995) asked participants to either rate the perceived effectiveness of the ILEX resistance training intervention as 'excellent,' 'good,' 'fair' or 'poor' which were rated respectively as 46%, 30%, 14% and 8%. Leggett et al. (1999) reported that all subscales of the SF36 form showed significant improvement in response to the ILEX resistance training intervention. In addition they asked participants to rate their outcome as either 'better,' 'same' or 'worse' which between the two centres ranged respectively from ~74% to ~82%, ~12% to ~24% and ~1% to 5%. Willemink et al. (2012) measured GPO at 12 and 24 weeks of their ILEX resistance training intervention as 1 = 'completely recovered,' 2 = 'much improved,' 3 = 'slightly improved,' 4 = 'no change,' 5 = 'slightly worsened,' 6 = 'much worsened,' and 7 = 'worse than ever.' The results respectively were rated 1 or 2 = 43.8%, 3 to 5 = 56.3%, and 5 to 7 = 0% at 12 weeks, and 1 or 2 = 50.0%, 3 to 5 = 37.6%, and 5 to 7 = 12.5% at 24 weeks.

Recently, international consensus has been offered on what is referred to as the 'Minimal Clinically Important Change' (MCIC) for changes in measures of perceived pain and

disability (Ostelo et al., 2008). The MCIC refers to the minimal change required in an outcome variable for it to have any meaningful impact upon a participant's perception of the overall outcome from an intervention. Thus it is usually considered with reference to the mean change found in a group for such a variable that has also reported a minimal positive perception of outcome in some form of GPO (De Vet et al., 2006; Kovacs et al., 2008). Ostelo et al. (2008) have suggested MCICs of 15mm for VAS, 10pts for ODI, 5pts for RMDQ or at least a 30% improvement from baseline. Considering these MCICs the studies reported here examining ILEX resistance training interventions consistently achieve these outcomes for VAS (Mooney et al., 1993; Park et al., 2000; Lee et al., 2000; Holmes et al., 1996; Steele et al., 2013^a; Choi et al., 2005; Smith et al., 2011; Bruce-Low et al., 2012; Stephan et al., 2011), ODI (Steele et al., 2013^a; Choi et al., 2005; Smith et al., 2011; Bruce-Low et al., 2012; Stephan et al., 2011; Carlson & MacKay, 2010), and RMDQ (Al-Obaidi et al., 2005; Willemink et al., 2012; Helmhout et al., 2004^a; 2008^b), with few exceptions (Mooney et al., 1993; Ju et al., 2012; Kim et al., 2010; Costa, 2010) where participants in these studies had very low baseline ODI and VAS scores which may account for the lack of MCIC. Al-Obaidi et al. (2005) have reported that pre-intervention characteristics including fear avoidance beliefs and initial pain intensity may affect whether MCICs are met through ILEX resistance training, suggesting higher scores in both these characteristics predict failure to meet MCIC. However, the intention to treat analysis used in this study included 6 participants who did not complete the intervention as not achieving the MCIC, though reasons for not completing the intervention are not reported.

A number of studies have also examined whether improvements in pain and disability produced through ILEX resistance training interventions are long-lasting. Nelson et al. (1995) followed up participants 1 year after and reported that 94% of participants who had previously reported a GPO of either 'good' or 'excellent' had maintained these outcomes. This occurred despite low adherence to a prescribed program of home-based exercises

during follow up (53%). Leggett et al. (1999) conducted 1 year follow ups in both centres used in their study reporting maintenance of positive outcomes on the SF36 from discharge to 1 year at both centres. Choi et al. (2005), however, in post lumbar discectomy participants showed that at 1 year follow up VAS was similar for both the group training using ILEX resistance training and also the non-training control group; however, the ILEX resistance training group produced a significantly greater reduction in pain post intervention thus benefiting from a longer period of time with minimal pain after surgery. Helmhout et al. (2004^a; 2008^b) and Harts et al. (2008) in randomised trials conducted 9 month and 16 week follow ups post 8 to 10 weeks of ILEX resistance training exercise 1 to 2x/week with samples of 81 and 65 participants respectively. They also reported maintenance of outcomes for pain and disability over the follow-up however a number of participants (84%) elected to continue with the ILEX resistance training intervention over this period.

Collectively a range of studies, including both prospective single arm trials and randomised controlled trials, suggest ILEX resistance training is effective in producing reductions in pain and disability that are significant, clinically meaningful and may also be long-lasting.. However, these studies have utilised varied applications of this exercise approach and thus examination of control of the specific resistance training variables (i.e. the dose of exercise; Mooney, 1992; 2007) is key to providing recommendations on the best means of employing ILEX resistance training in practice. Some have suggested following the American College of Sports Medicine's (Kraemer et al., 2002; American College of Sports Medicine, 2009) recommendations for resistance training prescription (Helmhout et al., 2008^a). However these have received criticism and alternative evidence based recommendations of resistance training to improve strength, endurance and hypertrophy have been recently reviewed and suggested (Carpinelli et al., 2004; Fisher et al., 2011; Fisher et al., 2013). Further, most studies examining recommendations for application of ILEX resistance training have been conducted in asymptomatic populations

(Pollock et al., 1989; Graves et al., 1990^b; Carpenter et al., 1991; Tucci et al., 1992). Though these support recent recommendations for an approach involving a single set of repetitions performed to momentary muscular failure using a load that permits ~8-12 repetitions before reaching failure, performed in a slow and controlled manner, at a frequency of around once per week to improve strength, endurance and hypertrophy (Carpinelli et al., 2004; Fisher et al., 2011; Fisher et al., 2013), whether training in this manner using ILEX resistance training is most efficacious for improving pain, disability or other outcomes in symptomatic participants is a different question. As such the next section will report research that has looked to clarify the manipulation of specific resistance training variables (intensity of effort, load/repetition range, repetition duration, volume, frequency and range of motion) using ILEX resistance training so as to offer recommendations for its application in symptomatic populations.

2.4.4.2 Manipulation of Resistance Training Variables for use of ILEX Resistance Training

Two studies have examined the effect of altering 'intensity' of ILEX resistance training using ILEX (Helmhout et al., 2004^a; Harts et al., 2008) comparing 'high intensity training' (HIT) with 'low intensity training' (LIT) (Helmhout et al., 2004^a) and also with a waiting list control group (Harts et al., 2008) reporting no difference between groups for improvement in disability (RDMQ), or overall outcome (SF36 and GPOs) for HIT and LIT (Helmhout et al., 2004^a), and or between HIT, LIT and a waiting list control (Harts et al., 2008). However, unfortunately these studies were not appropriately designed and controlled to examine the effects of 'intensity' and have been recently commented upon (Steele, 2013^b). In addition more appropriate definition and use of the term 'intensity' in resistance exercise has been suggested (Fisher et al., 2011; Fisher et al., 2013; Steele, 2013^b; Fisher & Smith, 2012). Recent proposals (Steele, 2013^b) define that '*intensity refers to the degree or magnitude of a measurable characteristic or variable*' and thus cannot specifically be considered to refer to a particular variable (e.g. *load* or *effort* as is most common). Comparison of load requires control of effort by having participants train to

momentary muscular failure (MMF; Steele, 2013^b). Training for the HIT group in the first study (Helmhout et al., 2004^a) used 35% of their max ILEX strength, whereas the LIT group used 20%. In the second study (Harts et al., 2008), load was increased for the HIT group to 50% of their maximal lumbar extension strength whilst keeping the LIT group's training the same as previously. In neither study did the participants train to MMF.

Although intensity of *load* differed, it is impossible to know the degree to which *effort* also differed between HIT and LIT (Helmhout et al., 2004^a; Harts et al., 2008). Effort increases with increased load assuming all other variables are constant, yet the loads used and the degree of difference between HIT and LIT was small (HIT used 35%/50% of max strength, LIT used 20% of max strength). In fact the LIT group may have trained at a relative load similar to the HIT group as the author's note even the lowest possible load the ILEX device could not permit 20% in some participants (Helmhout et al., 2004^a). Considering typical repetitions ranges possible at different relative loads (Hoeger et al., 1990; Shimano et al., 2006), and the repetitions ranges used within these studies, both groups likely trained at similarly low effort. Thus lack of significant differences between groups is unsurprising. Further, HIT and LIT were presented to the participants as *"potentially equally effective for the lower back while targeting different aspects: strength in the HIT group versus mobility in the LIT group"* (pp 540; Helmhout et al., 2004^a) thus it is unsurprising that the HIT group made greater improvements in strength whereas the LIT group made greater improvements in TSK reflecting fear of movement. Despite the relatively low effort approach used by both HIT and LIT, the HIT group likely trained at a marginally higher effort and most outcomes showed a trend towards greater improvement in this group (Helmhout et al., 2004^a; Harts et al., 2008). That intensity of effort may be an important factor to consider in determining the effectiveness of ILEX resistance training has recently been noted (Steele et al., 2013^b). Other studies already mentioned in which participants have completed repetitions to MMF have shown significant improvements in all outcomes compared to non-training control groups (Steele et al., 2013^a; Smith et al.,

2011; Bruce-Low et al., 2012; Risch et al., 1993; Nelson et al., 1995) in contrast to the results of the waiting list control group comparison by Harts et al. (2008). Although increased load increases effort when repetitions performed are matched, no studies have directly examined the effect of different loads independently on clinical outcomes in CLBP whilst controlling for other variables. Neither have any studies directly compared differing repetition durations nor different set volumes in symptomatic participants.

Frequency of training has varied in studies of ILEX resistance training utilising either a 2x/week training frequency or a mixed training frequency of 2x/week for the first 2 to 4 weeks followed by training 1x/week for the remainder of the intervention. Kim et al. (2010) examined 40 participants recovering from lumbar discectomy training 2x/week, 1x/week, 1x/2weeks, or a non-training control. After surgery participants completed 12 weeks of training using ILEX resistance training at a frequency of 2x/week. Participants were then tested for lumbar extension strength, ODI and VAS before then being randomised into a group training 2x/week, 1x/week, 1x/2weeks, or a non-training control. The group training 1x/2weeks did not significantly improve either ODI or VAS. ODI improved significantly in both the 1x/week and the 2x/week groups whereas VAS only significantly improved in the 2 x/week groups. However, both VAS and ODI were very low when first measured after surgery and the initial 12 week training (0.9cm to 1.0cm and 10.4pts to 10.8pts respectively). Before surgery participants' VAS scores ranged from 7.7cm to 8.7cm and ODI from 83.8pts to 85.2pts indicating improvement from before surgery to the first measurement of these variables. However, during the time between these two measurements both surgery *and* 12 weeks of initial ILEX resistance training was performed it is unclear as to what degree either exerted these improvements. Bruce-Low et al. (2012) examined the effect of either 1x/week or 2x/week ILEX resistance training over a 12 week intervention upon VAS and ODI. They reported no significant differences between improvements in VAS or ODI for either 1x/week or 2x/week training.

Steele et al. (2013^a) recently examined the effects of manipulation of range of motion (ROM) during ILEX resistance training comparing full ROM to limited ROM (performed using only the mid 50% of the participants full ROM) training over 12 weeks. They reported no significant differences between improvements in lumbar extension strength across the full ROM in agreement with previous literature in asymptomatic participants (Graves et al., 1992^b). In addition there were no significant differences in improvements for VAS and ODI when training either using full or limited ROM ILEX resistance training.

Despite the lack of controlled research examining clinical outcomes in response to different load, set volumes and repetition durations, collectively research suggests that low frequency (1x/week) yet high effort (to momentary muscular failure) ILEX resistance training performed through either a full or limited ROM elicits can be recommended for best improvements in pain and disability. Though research indicates positive outcomes from ILEX resistance training and allows some specification of recommendations for achieving such outcomes, the question of its efficacy in comparison to other specific exercise approaches and alongside other co-interventions remains. The next section will report studies of different specific exercise approaches compared with ILEX resistance training in addition to its efficacy as a single intervention or part of multiple interventions.

2.4.4.3 Studies of ILEX Resistance Training and other Specific Exercise Approaches

Randomised controlled trials using ILEX resistance training with symptomatic participants appear to have only been conducted in comparison to floor/stability ball exercise approaches, and other TEX resistance machines. Udermann et al. (2004) reported no differences between 4 weeks of McKenzie exercise with and without ILEX resistance training 1x/week on 6 significantly improved subscales of the SF36 including pain in a sample of 18 participants. Helmhout et al. (2008^b) also reported no differences between a regular physiotherapy group and a group performing isolated lumbar extension resistance training using ILEX resistance training over 10 weeks and over 6 and 12 month follow-

ups. The physiotherapy group performed a variety of treatments with the physiotherapist including 65% of activities as exercise (i.e. trunk and leg strengthening - though physiotherapists were instructed to not use the specific lumbar extension device - core stability exercises, stretching and specific McKenzie exercise), 25% constituted aerobic activity, 10% instruction and advice, and less than 1% as passive modalities. However, one participant included in the physiotherapy group undertook ILEX resistance training and 2 of the 6 centres used during the study reported utilising the ILEX resistance training device despite being instructed not to for the physiotherapy group. Participants in the physiotherapy group that also received ILEX resistance training were included in analysis despite the co-intervention whereas two participants from the group exclusively training on the ILEX resistance training machine who also accidentally received a manual therapy co-intervention were excluded from analysis. The selectivity of participant inclusion for analysis is unclear as the authors reported following 'intention to treat' principles.

Smith et al. (2011) conducted a randomised controlled trial involving two groups performing a 12 week training intervention 1x/week and a non-training control group. The two training groups performed exercise using an ILEX resistance training device, however, one group trained with the restraints tightened as per the manufacturer's recommendations (thus providing ILEX) and the other group trained without the use of the restraints. The results showed that only the group training with use of the restraints (i.e. ILEX) improved in any of the outcomes measured which included lumbar extension strength, VAS and ODI.

Many of the studies that have utilised ILEX resistance training and reported that its effectiveness have used it alongside numerous co-interventions thus rendering it impossible to definitively conclude that the effective part of the intervention is indeed the inclusion of ILEX resistance training. For example, many studies have included co-interventions including; other forms of resistance training exercise (including machines

and free weights), aerobic exercise using ergometers (i.e. cycle, treadmill etc.), and also behavioural and lifting education (Mooney et al., 1993; Choi et al., 2005; Ju et al., 2012; Stephan et al., 2011; Nelson et al., 1995; Leggett et al., 1999; Costa, 2010; Udermann et al., 2004; Vincent et al., 2014). Other studies however have examined the use of ILEX resistance training as a single intervention (Park et al., 2000; Lee et al., 2000; Holmes et al., 1996; Steele et al., 2013^a; Smith et al., 2011; Bruce-Low et al., 2012; Kim et al., 2010; Risch et al., 1993; Carlson & MacKay, 2010; Al-Obaidi et al., 2005; Willemink et al., 2012; Helmhout et al., 2004^a; Harts et al., 2008; Udermann et al., 2004; Vincent et al., 2014). The results of both studies of ILEX resistance training as a single or co-intervention suggest similar efficacy between both approaches. Interventions using ILEX resistance training alongside co-interventions have shown improvements of approximately ~30% to ~50% gains in lumbar extension strength, ~26% to ~69% improvement in pain using either SF36 or VAS (~15mm to ~55mm), and ~17% to ~30% improvement in ODI score (2.21pts to 5.33pts), compared with studies of ILEX resistance training as a single intervention reporting ~20% to ~55% gains in lumbar extension strength, ~55% improvement in pain measured through VAS (~16mm to ~21mm), ~30% to ~50% improvement in ODI score (~10pts to ~14pts), and ~16% improvement measured using the RMDQ. A randomised controlled trial by Vincent et al. (2014) has recently compared the use of ILEX resistance training as a single intervention with ILEX resistance training as part of a full body machine based resistance training intervention in addition to a control group undergoing standard care (including bodyweight resistance exercises, dietary information and information about back pain) in 49 obese participants with CLBP. They reported that improvements in ODI, RMDQ and pain catastrophising were significantly greater in the full body training group compared with the single ILEX resistance training group. However, they only report group x time effects and do not report *p* values for pairwise comparisons were the changes reported for ODI qualitatively appear greater for the full body group (-11.4pts) compared with ILEX resistance training (-6pts) and controls (-1.5pts). Though results for the RMDQ and for lumbar extension strength respectively suggested greater

improvements both the full body group (-4.7pts and 40Nm) and control group (-2.1pts and 35Nm) compared to the ILEX resistance training group (-1.1pts and 23Nm) suggesting the manipulation of resistance training variables in the ILEX resistance training intervention (e.g. they did not train to MMF) may have been insufficient to address lumbar extensor deconditioning in these participants.

2.4.5 Discussion

Three areas were considered for the purposes of this review; 1) ILEX resistance training's efficacy upon perceived pain, disability and GPOs including the clinical meaningfulness of these outcomes in CLBP, 2) the manipulation of ILEX resistance training variables for best outcome to provide recommendations for clinical prescription, 3) and the comparison of ILEX resistance training and other specific exercise approaches, including use of ILEX resistance training as part of a multiple or single intervention approach. The studies reviewed under these areas demonstrate that interventions using ILEX resistance training consistently produce significant improvements in both pain and disability which consistently meet MCICs. For practitioners considering the implementation of ILEX resistance training when working with persons suffering from CLBP evidence suggests that a low frequency (1x/week) yet high intensity of effort (to momentary muscular failure) approach using either full or limited range of motion ILEX resistance training is most effective. There is a lack of studies examining with appropriate control the impact of manipulating different load, set volumes and repetition duration thus prudence suggests following recent evidence based recommendations regarding these variables for resistance training may be sensible (Carpinelli et al., 2004; Fisher et al., 2011; Fisher et al., 2013). Further, comparison with other specific exercise approaches has not been tested as rigorously as is desired in some studies due to short duration of intervention (Udermann et al., 2004) in addition to comparisons being confounded by both groups using ILEX resistance training (Helmhout et al., 2008^b). However, one study suggests ILEX resistance training may be better than other specific exercise approaches (Smith et

al., 2011) and studies suggest similar efficacy whether used as a single intervention or alongside co-interventions.

The nature of exercise performed using ILEX resistance training allows for an accurate quantification of the dose provided and specific application of this dose to an isolated area. In addition to this, the testing features of some ILEX resistance training devices allow accurate quantification of treatment progress. Finally, ILEX resistance training is a time efficient strategy for tackling CLBP (Helmhout et al., 2004^b). ILEX resistance training sessions require at least ~50% less time compared to regular physical therapy (Helmhout et al., 2008^b). A recent analysis suggests that greater benefit may occur with a greater frequency of exercise sessions (an additional eight sessions required to improve VAS scores by 1mm compared to controls [Ferreira et al., 2010]). ILEX resistance training specifically however is apparently very effective with only a single weekly session with no further benefit from additional sessions (Bruce-Low et al., 2012). It seems clear also that ILEX resistance training is just as effective as an individual treatment approach (Park et al., 2000; Lee et al., 2000; Holmes et al., 1996; Steele et al., 2013^a; Smith et al., 2011; Bruce-Low et al., 2012; Kim et al., 2010; Risch et al., 1993; Carlson & MacKay, 2010; Al-Obaidi et al., 2005; Willemink et al., 2012; Helmhout et al., 2004^a; Harts et al., 2008; Udermann et al., 2004; Vincent et al., 2014) and that the benefits can occur from as little as one session per week taking approximately 10-15 minutes with only 1-2 minutes of that comprising exercise. As one of the biggest economic losses through CLBP occurs due to work hours lost both through treatment and absenteeism, a workplace strengthening program (Mooney et al., 1993; Mooney et al., 1995; Matheson & Mooney, 2006; Dreisinger, 2000) using ILEX resistance training could be an effective occupational approach.

Mooney et al. (1995) demonstrated that the use of a rehabilitation protocol using ILEX resistance training in a strip mining facility with higher than average injury rates resulted in

significantly reduced injuries and a reduction of workers compensation costs from \$14,430 per month to \$380 per month. In addition Matheson and Mooney (2006) report the results of a study (Dreisinger, 2000) conducted within the airline industry utilising an ILEX resistance training program with 622 workers and 2937 control workers. Back injuries in the exercise group were 5.7 (<1%) per year compared to 179 (~6%) per year in the control group. A difference in costs was also noted, with cost of back injuries at \$206 in the exercise group and \$4,883 in the control group. Initial return to work also is considerably higher in post lumbar discectomy patients undergoing ILEX resistance training compared to home-exercise based controls (87% ILEX compared to 25% controls [Choi et al., 2005]). In those off work due to CLBP related complaints (~73 days off work) initial return to work following ILEX resistance training is around 72% (Nelson et al., 1995). Nelson et al. (1999) also showed that the use of a rehabilitation program using ILEX resistance training for those with LBP who had originally been referred for spinal surgery resulted in only 7% of the participants requiring the expensive procedure. On average the cost of ILEX resistance training program in this study was \$1950 compared to average total surgical costs ranging from \$60,304 - \$168,732. Large scale studies (Nelson et al., 1995; Leggett et al., 1999) with one year follow ups have also shown that direct health care costs may be reduced as those rehabilitated using ILEX resistance training were significantly less likely to re-utilise the general health care system. It should be noted that health care re-use due to ineffective treatment is one of the most significant contributors to total costs of LBP (Carpenter & Nelson, 1999). Thus it seems that in terms of costs ILEX resistance training perhaps offers an effective solution. Yet there are also other benefits to the specific exercise approach using ILEX resistance training.

The use of progressive specific resistance exercise in treating CLBP appears relatively uncommon at present, and the use of ILEX resistance training specifically even less so. For example, in the UK, according to one ILEX device company website (MedXonline.com), there are only 5 facilities with access to their ILEX device (though the

authors of this manuscript are aware of two others). Compared with the availability of their device in the United States there is quite a difference. Within Los Angeles alone there are at least 49 facilities each providing access to an ILEX device. If this is representative of other ILEX devices then on the whole availability seems limited in comparison with other specific exercise approaches. The relatively little access to the equipment despite evidence supporting its use, and the current burden of CLBP, might be explained by concerns expressed regarding the initial cost of purchasing such equipment (Smith et al., 2008) and depreciation costs of materials (Helmhout et al., 2004^b). However, when weighed against the costs to taxpayers and employers incurred by LBP (as noted in the introduction chapter), the cost of ILEX device purchase is paltry (Smith et al., 2008). The use of ILEX resistance training can further help to alleviate high costs involved with surgery (Nelson et al., 1999), the direct cost of health care re-utilisation (Nelson et al., 1995; Leggett et al., 1999) and the indirect costs involved with loss of work hours and insurance claims (Mooney et al., 1995 Dreisinger, 2000). In addition there are a range of ILEX devices available commercially which range in price (e.g. Lumbar Extension Machine, MedX, Ocala, Florida; BackUp Dynamometer, Priority One Equipment, Grand Junction, Colorado; Lower Back Revival System, OriGENE Concepts BV, Delft, the Netherlands etc.). Some offer sophisticated testing options whereas others are purely for exercise use. Although sophisticated testing might be desirable in research it may be less of a concern to clinicians and so more 'low tech' options might be considered. The reliability of ILEX resistance training use in treatment between separate facilities has also been shown (Leggett et al., 1999) and this would suggest that if more health care facilities were to obtain ILEX devices the results gained from treatment would be consistent across facilities. The costs of ILEX resistance training should be weighed against the benefits (including reduction of treatment time through its minimal approach) when making decisions in this regard (Helmhout et al., 2004^b).

Despite the current body of research in this area there is scope for further research regarding ILEX to be conducted. There is a lack of rigorous research examining ILEX resistance exercise in comparison with other specific exercise approaches. Also, considering this, the extent of potential placebo effects, though difficult to examine in exercise based studies (Dvir, 2007), is an area regarding ILEX resistance training also requiring examination as it is noted that engagement in any type of exercise might offer some benefit through such means (Lederman, 2010; Steiger et al., 2012^a). CLBP is being considered more commonly as a multifactorial disorder with an array of symptoms and associations (National Research Council, 1998; National Research Council and Institute of Medicine, 2001). The use of ILEX resistance training however has yet to be considered in the wider scope of CLBP's multifactorial nature. Some suggest it may offer a range of treatment effects (Helmhout et al., 2004^b). Yet it is unknown whether it may also confer as yet unseen benefits to other aspects of physical function and symptoms associated with CLBP as might be deduced from speculations regarding the role of lumbar extensor deconditioning in low back pain and injury (Pollock et al., 1989; Jones, 1993; Carpenter & Nelson, 1999; Smith et al., 2008; Steele et al., 2014^a). Indeed, although a proposed mechanism of action is the specific strengthening of the lumbar extensor musculature that this type of treatment offers and there is some evidence to support a link between clinical improvements and strength improvements (Steele et al., 2013^a; Nelson et al., 1995; Steele et al., 2013^b), it is necessary to further examine the 'black box' of treatment mechanisms as this has recently been questioned (Willeminck et al., 2012; Lederman, 2010; Steiger et al., 2012^a). Lastly, as some have complained of the costs involved with specialised equipment such as ILEX devices, future research should look to the possibility of the effects of other specific exercise (i.e. those described by Mayer et al. [2008]) as a kind of 'maintenance' program that could be performed after an initial specific exercise program using ILEX resistance training so as to reduce participants reliance upon specialised equipment, supervision and locations.

2.4.6 Conclusion

In conclusion, the studies considered in this review suggest that an ILEX resistance training intervention of low frequency (1x/week) yet high intensity of effort (to momentary muscular failure) approach using either full or limited range of motion, either as a single approach alongside co-interventions, is effective in producing significant and clinically meaningful improvements in pain and disability for those with CLBP. However, due to lack of research, it is less clear as to whether these improvements are in fact greater than might be achieved through other specific exercises.

2.5 Synthesis and Overarching Rationale for the Thesis

The conclusions from the systematic reviews of previous sections in this Literature Review chapter can be summarised as follows;

- 1) Specific deconditioning of the lumbar extensor musculature (i.e. reduced strength/endurance, atrophy and excessive fatigability) may be causally implicated in low back injury and LBP.
- 2) Though many forms of exercise can condition the lumbar extensors it seems that evidence best supports the use of ILEX for such purpose.
- 3) Specifically addressing lumbar extensor deconditioning in LBP through use of ILEX appears to produce significant and clinically meaningful improvements in pain and disability.

If these conclusions are considered in the context of a multifactorial framework as noted in the Introduction chapter we might hypothesise that lumbar extensor deconditioning is perhaps implicated in many of the other symptoms and dysfunctions associated with LBP, and that perhaps addressing this deconditioning using ILEX might produce further beneficial outcomes in terms of function. Indeed the following could be considered the initial premise stemming from review of the literature and affecting the direction of research undertaken towards this thesis

- *Initial Premise* - Lumbar extensor deconditioning is potentially implicated as a cause of low back injury and LBP, may influence other associated physical symptoms, dysfunctions and pain causing mechanisms in LBP, and in the majority of cases of LBP and chronic LBP may be a predominant causative factor (though it is not implied to be causative in all cases). The corollary of this being that exercise aimed at addressing this (i.e. ILEX resistance training) is a justified approach to

preventing and treating LBP and chronic LBP and may affect other symptoms and dysfunctions yet to be examined.

This is an area of considerable significance and the results of this thesis may further add evidence either for or against the deconditioning hypothesis. However, considering the costs of LBP, also of importance are the potential implications of redirecting treatment to address this area (lumbar extensor deconditioning) using a single mode approach (Helmhout et al., 2004^b; 2008^a). ILEX is already evidenced as a cost effective approach, and, if it imparts improvement to other aspects of dysfunction, might have even greater merit in potentially reducing the economic, occupational and personal costs of LBP through reconsidering the need for expensive passive and multidisciplinary approaches; a topic of both economic and practical significance (Breen et al., 2006).

As such, and in light of the literature reviewed herein, three studies using ILEX will be conducted towards this thesis. The purpose of these studies being to examine the effect of ILEX when considering other dysfunctions associated with LBP and in improving other aspects of physical function of the lumbar spine. Specifically, the physical factors examined will be limited ROM, gait abnormality and disc health in non-specific chronic LBP participants¹⁸. The following sections of this chapter provide a review of the literature regarding these factors in LBP offering specific rationale for their study.

¹⁸ It should be noted that, though this thesis has chosen to focus upon and is limited to discussion of these three factors, the overarching rationale of the thesis justifies the pursuit of research examining the role of lumbar extensor deconditioning and use of ILEX in other symptoms and dysfunctions associated with LBP also. Indeed future studies should seek to utilise a multifactorial framework to examine other factors not investigated here.

2.6 Areas of Empirical Study

2.6.1 Range of Motion and Isolated Lumbar Extension Exercise in Chronic Low Back Pain¹⁹

One common associated dysfunction in LBP is limited gross sagittal ROM of the lumbar spine (Pearcy et al. 1985; Beattie et al. 1987; Holmes et al., 1996; Nelson et al., 1995). In addition, LBP participants often experience exacerbation of pain when moved to their fully flexed or extended positions (Donelson et al., 1991). Combined with associated deconditioning, rehabilitation using lumbar extensor resistance training is commonly prescribed, with authors suggesting use of a limited ROM (Graves et al. 1992^b; McGill, 2007) that can be progressed as a participants ROM improves (Graves et al. 1992^b).

As already presented, ILEX is significantly effective in improving ILEX strength, pain, and disability in chronic LBP participants. However, research to this point has focused upon full ROM exercise. It has been shown that healthy asymptomatic participants training using limited ROM ILEX (36°) achieved significant gains in strength through their full ROM (72°; Graves et al., 1992^b). Other research has examined limited ROM training using differing exercises and joint movements, both isolated and compound. Additional work by Graves et al. (1989) reported limited ROM isolated knee extension and also produced significant strength increases throughout the full ROM (Graves et al., 1989). Limited ROM bench press training can produce significant increases in full ROM strength also (Massey et al., 2004). However, further study reported that, although limited ROM training produced significant full ROM strength improvement, full ROM training produced significantly greater increases in full ROM bench press in women (Massey et al., 2005). More recently Pinto et al. (2012) reported that, although muscle thickness increases similarly from full and limited ROM elbow flexion, full ROM strength increased significantly more from full ROM training. Further supporting limited ROM training, McMahon et al.,

¹⁹ Note that the content discussed in section 3.1 “*Isolated Lumbar Extension Exercise in Chronic LBP: Comparison of Limited Range of Motion and Full range of Motion Lumbar Extension Exercise*”, and the results and discussion from section 5.1, have also in part been published as a peer review journal article with the author as first author included in the appendices; Steele et al., 2013. A Randomised Controlled trial of Limited Range of Motion Lumbar Extension Exercise in Chronic Low Back Pain. *Spine*. 38(15), pp 1245 – 1252.

(2014) examined the effects of knee ROM using free weights, resistance machines and bodyweight exercises for the lower body. Both training full and limited ROM groups significantly improved CSA at 8 weeks at all sites. Only one significant difference in favour of the full ROM group was found for CSA at 75% site at week 8.

Limited ROM exercise for both the lumbar extensors (Graves et al.1992^b) and the knee extensors (Graves et al. 1989) have demonstrated non significant trends towards greater gains in the trained ROM, suggesting there may be a specific interaction. Research looking to identify if angle specific strength training produces angle specific strength increases using isometric exercise did not support this (Knapik et al., 1983), However, measurements were only taken 20° either side of the angle trained so it is unknown whether strength improved over the full ROM (Knapik et al., 1983). Adaptations within a specific ROM may perhaps be predominantly neuromuscular as the muscle fibers attached to corresponding motor neurons do run the full anatomical length of the muscle. Indeed research has shown that although measured strength significantly increases at all angles after angle specific elbow flexor training, increase in maximal voluntary contraction is greater at the trained angle and associated with an increase in maximal integrated EMG (Thépaut-Mathieu et al. 1988). Collectively however, these studies suggest the possibility that limited ROM training produces improvements in muscular strength and hypertrophy across areas of the untrained ROM.

Limited ROM ILEX exercise may therefore have merit for those suffering with chronic LBP and limited ROM by improving full ROM strength as well as pain and disability. However, as Graves et al. (1992^b) only examined healthy asymptomatic individuals study into limited ROM ILEX exercise in symptomatic populations is justified to determine whether limited ROM training is indeed effective in this population.

2.6.2 Gait Variability and Isolated Lumbar Extension Exercise in Chronic Low Back Pain²⁰

A further association with LBP often reported is gait variability (Waddell et al., 1997; McGill, 2007; Norris, 2008). Average movement amplitudes of the trunk and pelvis in chronic LBP participants are usually not significantly different from asymptomatic participants (Vogt et al., 2001; Lamothe et al., 2006^a; Seay et al., 2011^a). However, chronic LBP participants do present differently in other aspects of lumbar spine movement, such as inability to adapt pelvis/trunk coordination phase differences during increases in walking velocity, and greater stride-to-stride variability of lumbar spine kinematics with respect to the pelvis. Healthy participants demonstrate relatively low stride-to-stride variability in lumbar kinematic patterns during both level and incline gait (Vogt et al., 1999). However, greater stride-to-stride variability at the lumbar spine in all movement planes (Vogt et al., 2001), greater frontal plane coordination variability of the pelvis and trunk (Lamothe et al., 2006^a; Seay et al. 2011^b) and more rigid transverse plane coordination variability of the pelvis and trunk (Lamothe et al., 2002; Lamothe et al., 2006^a; van der Hoorn et al., 2012) is reported in chronic LBP participants compared with healthy controls. These atypical patterns are combined with poorer erector spinae activity adaptability to unexpected perturbations (Lamothe et al., 2004), or walking velocity changes (Lamothe et al., 2006^b). In fact, the findings of numerous studies are suggestive of lumbar extensor dysfunction during gait in those with chronic LBP compared with asymptomatic controls (Arendt-Nielsen et al., 1996; Vogt et al., 2003; Lamothe et al., 2004; Lamothe et al., 2006^a; Lamothe et al., 2006^b). Hanada et al. (2011) also reported that where asymptomatic controls significantly activated their rectus abdominus and internal obliques more, symptomatic participants had significantly greater lumbar extensor activation. More recent work showed evidence of greater lumbar extensor activity in chronic LBP

²⁰ Note that the content discussed in section 3.2 “*Isolated Lumbar Extension Resistance Exercise Effects upon Gait Variability in Chronic Low Back Pain Participants*” has been presented in part by the author in a conference presentation; Steele et al., 2013. Isolated Lumbar Extension Resistance Training Reduces Lumbar Kinematic Variability During Gait in Chronic Low Back Pain Participants. *World Low Back & Pelvic Pain Congress*) which is available at: https://www.researchgate.net/publication/258241462_ISOLATED_LUMBAR_EXTENSION_RESISTANCE_EXERCISE_REDUCES_LUMBAR_KINEMATIC_VARIABILITY_DURING_GAIT_IN_CHRONIC_LOW_BACK_PAIN_PARTICIPANTS. Also, at the time of submission, this content is currently published in part with the remainder being considered under peer review for publication by the author; Steele et al. The Effects of Isolated Lumbar Extension Exercise on Lumbar Kinematic Pattern Variability during Gait in Chronic Low Back Pain. *PM & R*. Under review; Steele et al. Lumbar Kinematic Variability during Gait in Chronic Low Back Pain and Associations with Pain, Disability and Isolated Lumbar Extension Strength. *Clinical Biomechanics*. Published ahead of print.

participants compared with controls (van der Hulst et al., 2010^a), at a range walking velocities (van der Hulst et al., 2010^b), and that neither disability nor fear of movement is associated with this greater activity (van der Hulst et al., 2010^c).

The lumbar spine plays an important role in driving human bipedal gait (Gracovetsky, 1985). It is possible that greater activation of the lumbar extensors, and altered lumbar spine kinematics during gait in chronic LBP participants, are manifestations of lumbar extensor deconditioning. Deconditioning therefore may impact upon motor control strategies. Greater activation in the face of fatigue, due to deconditioning, could be a compensatory attempt to maintain control of the lumbar spine during gait. Hart et al. (2009) demonstrated that inducing fatigue in the lumbar extensors impacts lumbar kinematics during running gait of healthy participants and chronic LBP participants. Arjunan et al. (2010) also showed significantly greater lumbar extensor activity during running gait in chronic LBP participants. Indeed, prospective evidence supports lumbar extensor deconditioning as being a risk factor for low back injury and pain (Biering-Sorenson, 1984; Luoto et al., 1995; Salminen et al., 1995; Lee et al., 1999; Sjolie et al., 2001). Thus it may also be responsible for the development of the atypical gait associated with chronic LBP.

Exercise programs have been successful in improving gait variability in older individuals and improvement appears to be in part determined by gains in strength (Hausdorff et al., 2001). As noted however, specific lumbar extensor exercise is often used to address lumbar extensor deconditioning (Mayer et al., 2008) and thus may be valuable in addressing the associated lumbar spine kinematic gait variability also. Varied exercise based interventions (Pilates, trunk extensions, stability exercise, transverse abdominus exercise) elicit improvements in gait control in chronic LBP participants (Carpes et al., 2008; Tsao & Hodges, 2008; Da Fonseca et al., 2009). However, ILEX has been presented as a more specific means of conditioning the lumbar extensors. In addition,

recent work reported improvement in ILEX strength, resulting from a strengthening program, predicted improvement in gait endurance in chronic LBP participants (Vincent et al., 2013). The role of lumbar extensor deconditioning in gait control suggests ILEX may hold value for improving it. However, it has yet to be examined for its effects upon lumbar kinematics during gait specifically.

2.6.3 Intervertebral Disc Hydration and Isolated Lumbar Extension Exercise in Chronic Low Back Pain²¹

The intervertebral discs are a suspected source of pain in LBP and disc abnormalities are more common in symptomatic participants than asymptomatic ones (Endean et al., 2011; McNee et al., 2011; Shambrook et al., 2011). A common, potentially painful disc abnormality is disc herniation (Hollingworth et al., 1998). Disc herniation is thought to typically occur in younger, more hydrated discs (Adams & Hutton, 1985; Adams & Muir, 1976) whereas older degenerated discs are generally characterised by cracks (Goel et al., 1995). However, more recently it was shown that degenerated discs, with lower osmotic pressures and decreased annular stresses, enhance the opening of cracks in the anullus leading to herniation (Wognum et al., 2006). Videmann et al. (1995) documented that vertebral body osteophytes are associated with end plate irregularity and disc bulging. Yet osteophytes are generally accepted as secondary to disc and end pate trauma despite taking years to develop (McGill, 2007). Thus degenerative discs may be at greater risk of herniation.

Biochemical analysis of the changes involved in symptomatic degenerative discs compared to asymptomatic discs shows that significant metabolic abnormalities are present including; reduced glycosaminoglycans, dehydration, and reduced nucleas

²¹ Note that the content discussed in section 3.1 “*Intervertebral Disc Hydration and Isolated Lumbar Extension Exercise in Chronic Low Back Pain*”, and the results and discussion from section 5.1, have also in part been published as a peer review journal article with the author as first author; Steele et al., 2013. Can Specific Loading through Exercise Impart Healing or Regeneration of the Intervertebral Disc?. *The Spine Journal*. Accepted and in press. Also, at the time of submission, part of this content is currently being considered under peer review for publication as independent articles by the author; Steele et al. Determining the reliability of a custom built seated stadiometry set-up for measuring spinal height in participants with chronic low back pain. *Applied Ergonomics*. Under review.

pulposus pH (Kitano et al., 1993). Loss of disc hydration and height in particular is commonly considered indicative of pathological processes as opposed to being age related degeneration (Adams & Roughley, 2006; Griffith et al., 2007). Disc hydration is often measured via magnetic resonance imaging (MRI; Paaanen et al., 1994). However, this is expensive and not routinely available. Indirect measurement though can be obtained using seated stadiometry to measure spinal height.

A number of studies have used stadiometry, both standing and seated, to examine the effects of different variables upon spinal height. There is a well-documented effect of time of day (diurnal variation) upon stature (Reilly et al., 1984; Tyrell et al., 1985) similar in both standing and seated stadiometry, suggesting most stature loss comes from the spine (McGill et al., 1996). Using MRI, research confirms a diurnal loss in disc height to support this (Paaanen et al., 1994). Changes in stature have been used to examine the effects of acute loading patterns upon changes in spinal height also. Acute resistance type exercise elicits a reduction in spinal height (Wilby et al., 1987; McGill et al., 1996), as do acute plyometric drop jump and pendulum based exercises (Fowler et al., 1997). Changes in recovery postures, such as lying supine with or without hyperextension, have also been shown to elicit recovery of stature loss from loading (Magnusson et al., 1996; Healey et al., 2004; Kourtis et al., 2004). In turn, recovery of stature has been shown to be associated with recovery of disc height via MRI also (Kourtis et al., 2004).

Regular movement and exercise of the lumbar spine is suggested to reduce loss in disc hydration (Norris, 2008; Mooney et al. 2006; Mayer et al. 2008). Nelson et al. (1995) reported that reduction in pain after ILEX exercise was similar in all diagnosed conditions including degenerative disc disease. Concerns have been expressed regarding the safety of using exercise such as ILEX when considering disc health (McGill, 2007). However, reviews have suggested that, although disc degeneration can be affected negatively by loading, the potential for a “safe window” of disc loading that may stimulate optimal disc

health does exist (Stokes & Latridis, 2004; Chan et al. 2011). Indeed recently the available animal model research has been reviewed and found to suggest its biological plausibility (Steele et al., 2014^b). The studies reviewed suggested utilising a relatively high magnitude, short frequency and short duration dynamic loading to produce potentially regenerative effects upon the intervertebral disc (including improvements in disc proteoglycan content, matrix gene expression, rate of cell apoptosis and improved fluid flow and solute transport; Walsh & Lotz, 2004; Maclean et al., 2004; Ferguson et al., 2004; Maclean et al., 2005; Wang et al., 2007).

As ILEX allows quantification of load and specific application to the lumbar spine it is a suitable tool for examining the effect of controlled loading upon disc condition in chronic LBP participants. ILEX has been shown to produce successful rehabilitation outcomes in participants diagnosed with degenerative discs (Highland & Dreisinger, 1992; Nelson et al. 1995) in addition to participants undergoing lumbar discectomy for disc herniation (Choi et al., 2005). However no studies have quantified any change occurring in disc condition *in vivo*.

As noted, it is recommended that a heavy session of ILEX be performed 1x\week for optimal stimulation of muscular adaptation and a light 'mobilisation' session be performed in addition, which was thought to enhance disc hydration through pressure variation across the annulus (Jones, 1993; MedX Utilisation Steering Committee, 1995-1996). However, this has been shown to be unnecessary for optimal strength, pain and disability improvements in chronic LBP participants (Bruce-Low et al., 2012). In addition Walsh and Lotz (2004) reported that lower frequency and higher load compression is optimal for improvements in disc proteoglycan content *in vitro*. Thus it is of interest to examine the effect of infrequent yet heavy applied loading to the lumbar intervertebral discs through ILEX.

2.7 Summary of Literature Review

It is clear that LBP is a highly prevalent and costly multifactorial condition, with which there are numerous symptoms and dysfunctions associated, which may be potentially pain causing in their mechanisms, and representative of dysfunction in a number of areas. Although a number of possible causative and influencing factors exist, evidence from a triumvirate of research methods suggests lumbar extensor deconditioning is commonly associated with, and potentially causative in, LBP. Consideration of LBP using a multifactorial framework suggests many common associations may therefore result from lumbar extensor deconditioning. Because of the association with lumbar extensor deconditioning a specific exercise based approach may be justified and ILEX has been shown to be optimal for conditioning the lumbar extensors, in addition to producing significant and meaningful improvements in outcomes such as pain and disability. Further, it is similarly effective compared to other approaches, including surgery, especially through reduction in re-utilisation of the health-care system. ILEX is also a time efficient approach allowing valid and reliable quantification of objective improvements in the lumbar extensors to assess rehabilitation efficacy. The efficacy of ILEX in comparison to other rehabilitation approaches has not been tested as rigorously as desired and there is need for better designed trials comparing them. However there is good reason to believe that the specific approach that ILEX devices provide allows optimal functional and clinical improvements. In light of the literature reviewed herein, however, the author suggests further research identify whether treatment of lumbar extensor deconditioning in LBP through ILEX holds further relationships with associated dysfunctions in LBP.

The three specific factors (limited ROM, gait variability, intervertebral disc degeneration) considered within this overarching rationale that constitute the areas of empirical study conducted towards this thesis have been reviewed, justifying their investigation, and the following section now details the specific research questions and hypotheses to be examined.

2.8 Proposed Research Questions and Hypotheses

The following studies will be conducted to consider three research questions and test the corresponding proposed hypotheses based upon the extant literature:

- **Study 1** - Can ILEX resistance training through a limited ROM can produce strength gains throughout the full ROM in symptomatic chronic LBP participants?
 - **Hypothesis 1** - Limited ROM ILEX resistance training will produce full ROM strength improvement in chronic LBP participants in addition to improving ROM and reducing pain and disability with no difference between limited or full ROM exercise.
- **Study 2** - Can ILEX resistance training effect gait variability in chronic LBP participants?
 - **Hypothesis 2** - ILEX resistance training will produce reductions in gait variability in chronic LBP participants.
- **Study 3** - Can ILEX resistance training effect inter-vertebral disc hydration, measured via seated stadiometry, in chronic LBP participants?
 - **Hypothesis 3** - ILEX resistance training will produce improvements in intervertebral disc hydration measured indirectly via seated stadiometry.

3. METHODS

The following sections detail the general methods applied to this area of research including the equipment and materials utilised. In addition specific methods and materials relevant to the examination of the specific areas examined within this thesis are also noted in their respective sub sections. For clarity, the respective areas shall be referred to as study 1, 2 and 3 and correspond to the research questions noted at the end of the Literature Review chapter.

3.1 Study Design

3.1.1 Study Design; Study 1 and Study 2

For the purposes of study 1 and study 2 a randomised controlled trial design was adopted with two experimental groups and a control group. In study 2 the two experimental groups from the wider investigation (FullROM & LimROM) were combined to form a single experimental group who had performed training using ILEX. The studies were approved by the NHS National Research Ethics Service, Southampton & South West Hampshire Research Ethics Committee B (REC Reference: 11/H0504/9) and the Centre for Health, Exercise and Sport Science ethics committee at Southampton Solent University (SSU) and within the Sport and Exercise Science Laboratories at SSU.²²

3.1.2 Study Design; Study 3

Study 3 followed a quasi-experimental wait-list controlled design with all participants undergoing pre testing (T1) followed by an initial 12 week control period, before being retested (T2) and then beginning the 12 week experimental period. Participants were post tested once the experimental period had finished (T3). This study was approved by the ethics committee at SSU and also conducted within the Sport and Exercise Science Laboratories at SSU.²³

²² See appendix 7.3

²³ See appendix 7.3

3.2 Participants

An *a priori* power analysis (Figure 5) of previous research with chronic LBP participants (Choi et al., 2005) was conducted to determine participant numbers (n) using a treatment effect size, calculated using Cohen's *d* (Cohen, 1992), of 1.48 for the ILEX strength measured using the MedX Lumbar Extension Machine. Participant numbers were calculated using equations from Whitley and Ball (2002). These calculations showed that each group within the studies conducted required 7 participants to meet the required power of 0.8 at an alpha value of $p \leq 0.05$.

$$ES = \frac{107.18^a - 162.57^b}{37.28^c}$$

$$ES = 1.48$$

$$n = \frac{2}{1.48^2} \times 7.9$$

$$n = 7.2$$

Figure 5. Power Analysis to determine participant numbers: ^a Mean pre-test training group, ^b Mean post-test training group, ^c Control group standard deviation; data from Choi et al. (2005)

Thus, the studies were considered to be adequately powered. In addition, and relevant to study 2, this number of participants combined with 5 kinematic trials per participant is considered sufficient for achieving adequate statistical power in kinematic research (Bates et al., 1992).

General inclusion criteria for all three studies were as follows: participants were ≤ 18 years old, suffered from non-specific low back pain having lasted longer than 12 weeks (Frymoyer, 1988) and had no medical condition for which resistance training would be contraindicated. Exclusion criteria²⁴ for all three studies were as follows: participants must

²⁴ These exclusion criteria were determined by the consulting Chiropractor who conducted participant screening, Dr Neil Osborne, Head of Clinic at the Anglo European College of Chiropractic, through consultation with additional experts within

have no medical condition for which movement therapy would be contraindicated. These included: acute (not re-occurring) low back injury occurring within the last 12 weeks, pregnancy, evidence of sciatic nerve root compression (sciatica), leg pain radiating to below the knee, paraesthesia (tingling or numbness), current tension sign, lower limb motor deficit, current disc herniation, previous vertebral fractures or other major structural abnormalities. All participants were cleared prior to involvement in the study by either their General Practitioner or the Chiropractor in the research group and provided written informed consent (copies are included in Appendix 7.4).

3.2.1 Participants; Study 1 and Study 2

Thirty eight participants (males $n = 21$, females $n = 17$) were initially identified and recruited by posters, group email and word of mouth from SSU and the surrounding locality. Direct referral was also provided from a local private chiropractor in addition to posters in their practice.

Figure 6 shows a CONSORT diagram highlighting the participant numbers for enrolment, allocation, follow-up and analysis stages for study 1²⁵. After initial drop outs thirty one participants were randomised using a randomisation program (Research Randomizer vs. 3.0) to one of three participant groups; a full ROM training group (FullROM; $n = 12$), a limited ROM training group (training using the mid 50% of their ROM; LimROM; $n = 10$), and a control group ($n = 9$) who did not train but continued with any treatment or intervention they were currently undertaking.

the area including Dr Stuart McGill, Professor at University of Waterloo, Canada, Dr Andry Vleeming, Professor at University of Ghent, Belgium, and Dr Michael Schneider, Assistant Professor at University of Pittsburgh, United States of America

²⁵ Note that two CONSORT diagrams are presented for study 1 and study 2 despite them both being conducted in tandem using the same participants. This is due to some participants not completing, or their data being unavailable for one study but not the other. Thus these are detailed separately.

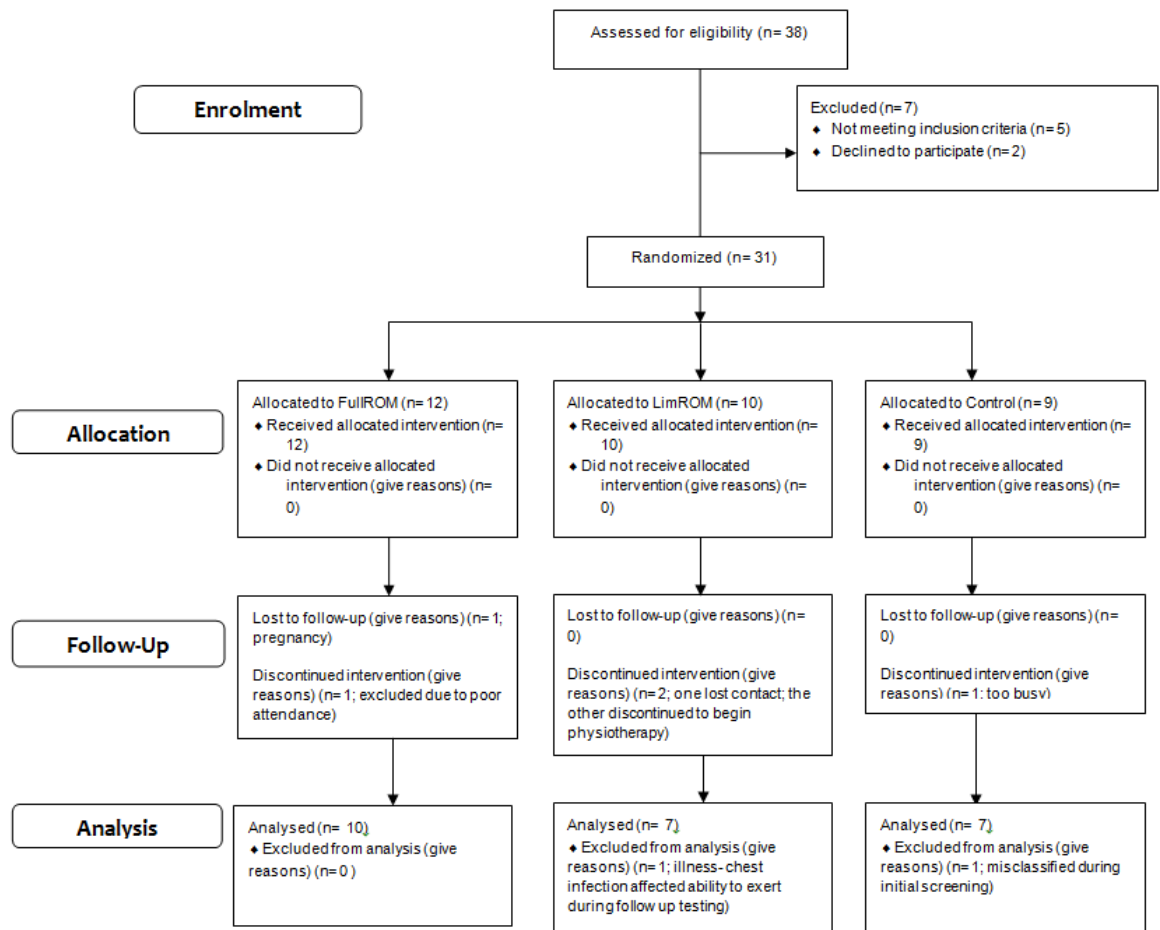


Figure 6. CONSORT diagram to illustrate participant numbers for enrolment, allocation, follow-up and analysis stages for study 1

Figure 7 shows a CONSORT diagram highlighting the participant numbers for enrolment, allocation, follow-up and analysis stages for study 2. As noted, the two experimental groups were combined for analysis in this particular study.

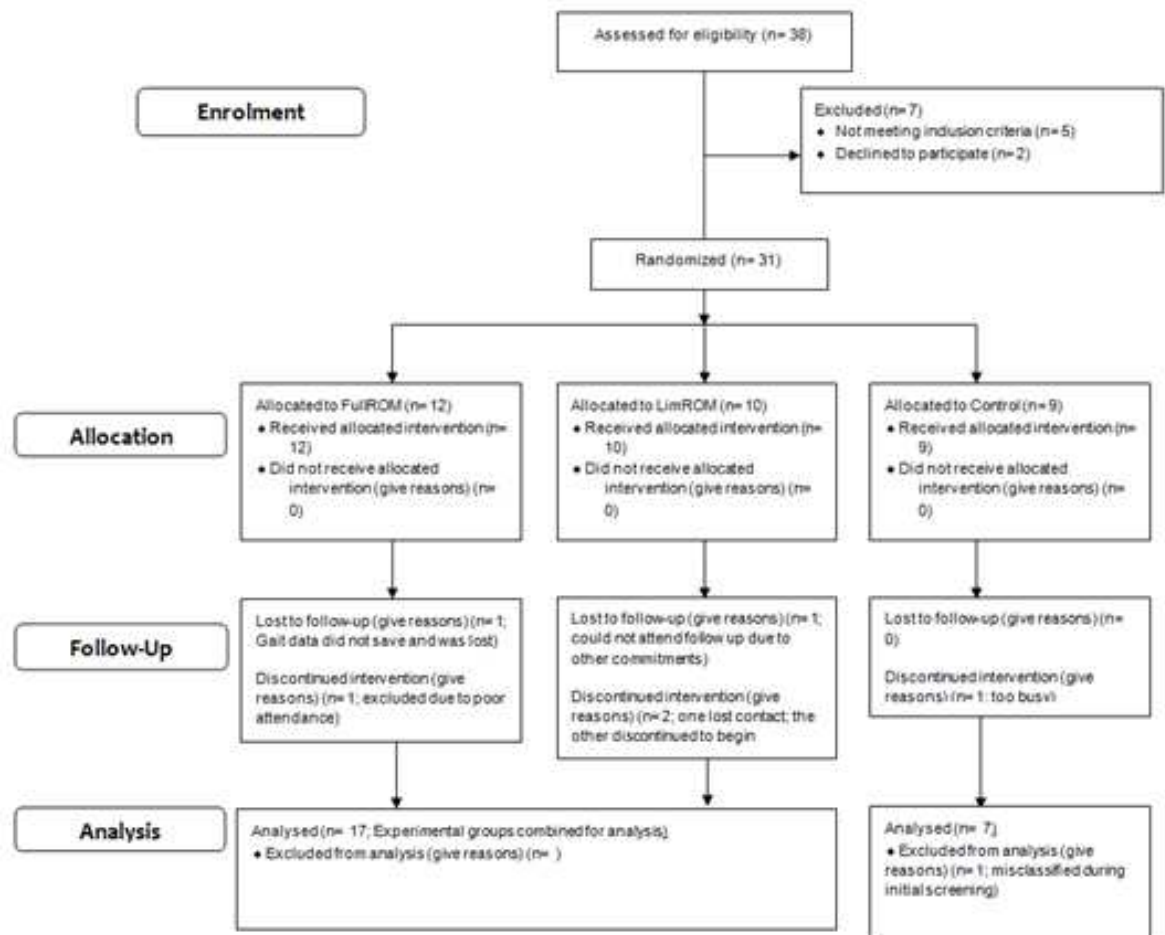


Figure 7. CONSORT diagram to illustrate participant numbers for enrolment, allocation, follow-up and analysis stages for study 2

3.2.2 Participants; Study 3

Seventeen participants (males n = 9, females n = 8) were initially identified and recruited by posters, group email and word of mouth from SSU and the surrounding locality. Direct referral was also provided from a local private chiropractor in addition to posters in their practice. After pre-testing participants underwent a 12 week control period where they were instructed to continue with their daily activities as normal and any treatment or intervention they were currently undertaking. After completion of this 12 week period participants were re-tested and then underwent a 12 week ILEX exercise training intervention. Figure 8 shows the flow of participants through the study.

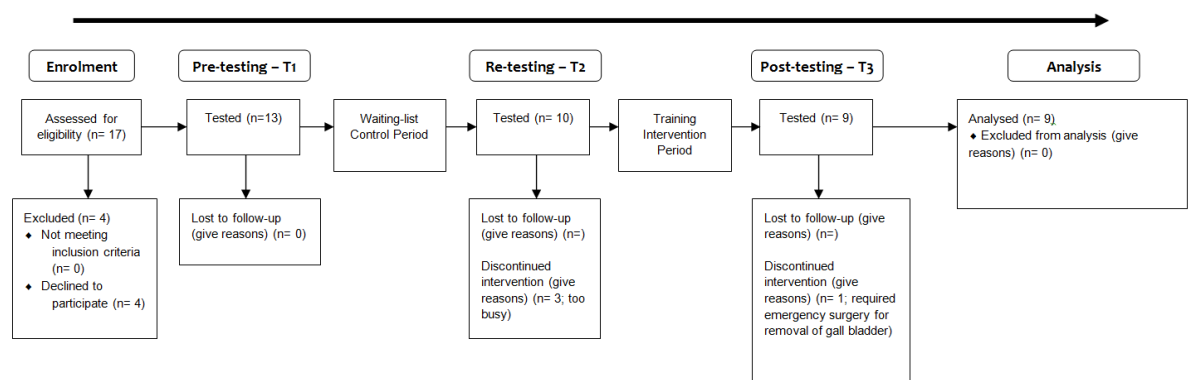


Figure 8. Flow of participants through study 3

3.3 Equipment

The following are the general measures and equipment used in all studies. Participants' stature was measured using a stadiometer (Holtan Ltd, Crymych, Dyfed) and body mass measured using scales (SECA, Germany) and Body Mass Index (BMI) calculated. Isometric strength testing, ROM and training were performed using the MedX Lumbar Extension Machine (Medx Corporation, Ocala, Florida; Figure 4). The ILEX machine has been shown to be reliable in assessing isometric strength at repeated angles in asymptomatic ($r = 0.81$ to 0.97 ; Graves et al, 1990^a) and symptomatic participants ($r = 0.57$ to 0.93 ; Robinson et al. 1992^a), and valid in measurement (Pollock et al. 1991; Inanami, 1991). Pain was measured using a 100mm point visual analogue scale (VAS; Ogon et al. 1996), and disability measured using the revised Oswestry disability index (ODI; Fairbank et al. 1980).

3.3.1 Equipment; Study 1 and Study 2

In study 1 and study 2 standing ROM was also measured using the modified Schober's test in both flexion (SchFlex; Gill et al. 1988) and extension (SchExt; Beattie et al. 1987). Gait kinematic variables were captured at 500hz using a 10 MX T20 camera three dimensional motion capture system (Vicon, Oxford) and analysed using both Vicon Nexus software version 1.4.116 (Vicon, Oxford), MATLAB version R2012a (MathWorks, Cambridge) and Microsoft Excel version 2010 (Microsoft, Reading).

3.3.2 Equipment; Study 3

In study 3 participants' seated stature (for indirect determination of spinal height) was also measured using a wall mounted stadiometer (Holtan Ltd, Crymych, Dyfed). Details of seated stature measures are detailed below. A customised wooden seat in addition to custom built wall mounted adjustable postural rods (Figure 9; SSU, Southampton) were used with the wall mounted stadiometer for seated stature measurements to ensure participants adopted the same posture for each retest trial. The back rest of the wooden seat was removed and replaced with a short solid wooden backboard for positioning of the sacral crest and a similar wooden board placed across the rear of the seat's legs to position and secure it against the foot board of the wall mounted stadiometer. The placement of the postural rods mounted to the wall was noted as the vertical distance measured from the floor to the top of the mount and was also traced as a line on the wall with the participants ID noted next to it. This was to ensure that the vertical position of the postural rods was the same for each test. The horizontal distance of the postural rods was ensured by measuring and recording the horizontal distance of the rod from its base to the left most insertion of the rod clamp. Spirit level vials were attached to each of the postural rods also to ensure that they were level in the coronal plane when setting up and taking measurements. Figure 10 shows a schematic depiction of the set-up for measurement of seated stadiometry.



Figure 9. Custom built wall mounted adjustable postural rods.

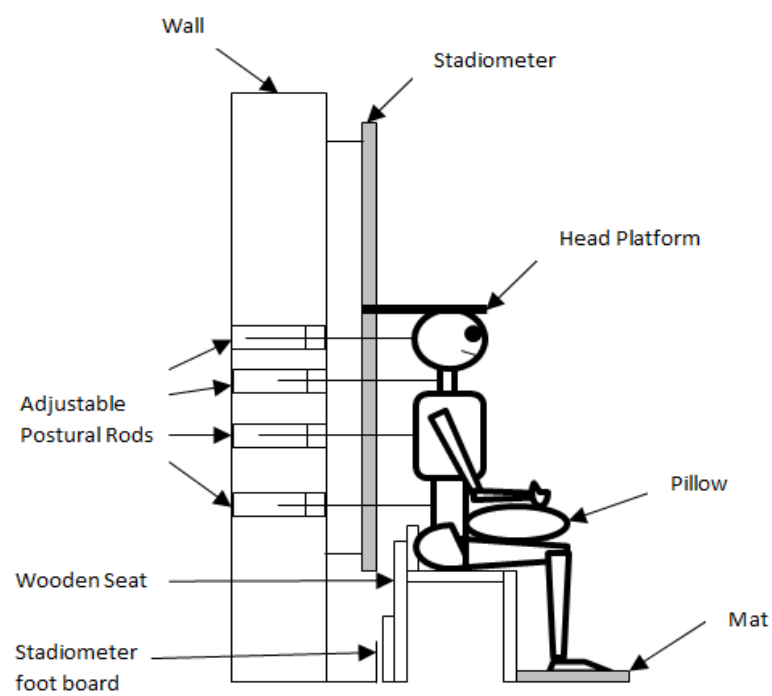


Figure 10. Schematic of seated stadiometry setup.

3.4 Participant Testing

3.4.1 Participant Testing; Study 1 and Study 2

Isometric ILEX strength was tested twice, on separate days (at least 72 hours apart in order to avoid the effects of residual fatigue or soreness) both before and after the intervention. Each test using the lumbar extension machine involved maximal voluntary isometric contractions at various angles through the participant's full ROM. Details of the full test protocol using the lumbar extension machine and details of the restraint mechanisms have been documented previously elsewhere (Graves et al., 1990^a) and the equipment manufacturer's operation instructions for conducting testing are included in Appendix 7.4. During the first and second to last visit to the laboratory, participants were required to complete the VAS and the ODI, and also to have their standing ROM measured using the modified Schober's test. Gait data were collected using the Vicon system during the third visit to the laboratory, and also during the participant's final visit to the laboratory after the intervention period.

3.4.1.1 Three Dimensional Motion Analyses

Due to the lumbar spine's capacity to rotate about three orthogonal axes, a three dimensional approach was used for data collection. Ten cameras were set up and angled in a manner so as to reduce hidden spots that might obscure data collection. Figure 11 shows the setup of the cameras relative to the participant during walking trials as viewed using the Nexus software. The cameras identified reflective markers attached to the participant and output three dimensional coordinates for each marker. Data were recorded for 5 walking trials both pre and post intervention. Participants walked barefoot from one end of a marked runway to the other that was 8 metres in length at their free walking speed. The first full gait cycle captured where the participants entered the calibration volume during each walking trial was used.

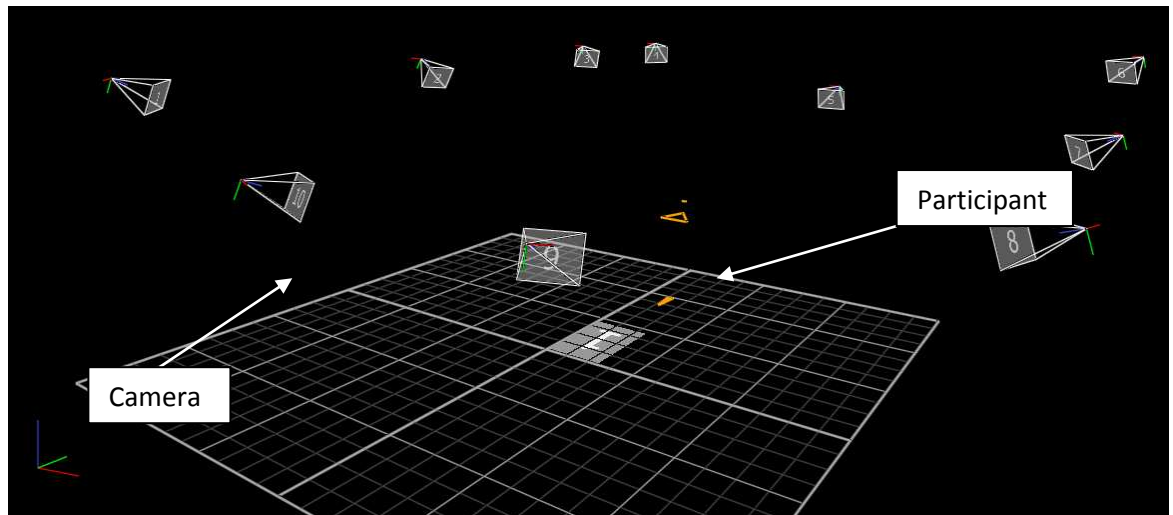


Figure 11. Three dimensional motion camera set up

3.4.1.2 Biomechanical Model

The body of interest for the current study was the lumbar spine considered from S1 to T12 relative to the pelvis. For the purpose of analysis the lumbar spine was modelled as a rigid segment. The reasoning for not considering intervertebral segment movements was due to the small segments ranging from S2 to T10 always bending laterally toward the support leg with little variation between segments (Syczewska et al., 1999). Lumbar spine data were collected through three axes using the same model previously described by Schache et al. (2002^a; 2002^b), which has been shown to have high overall repeatability of angular parameters (Schache et al., 2002^b).

3.4.1.3 Marker Set Up

All markers were placed by the same investigator for all gait trials. Reflective markers (Figure 12) were placed over anatomical landmarks on the pelvis at both anterior superior iliac spines (ASIS) and at the midpoint of the posterior superior iliac spine (PSIS) using double sided adhesive tape. Reflective markers were also used upon a thoraco-lumbar marker cluster similar to that used by Schache et al., (2002^a; 2002^b). As with the biomechanical model, this marker set up has been previously described elsewhere (Schache et al., 2002^a; 2002^b). The only alteration in this present study was the use of a

flexibly-based wand marker for the thoraco-lumbar cluster. Two additional markers were secured equidistant either side of the midpoint of the wand markers base. This was placed in the same position over the 12th thoracic spinous process with the mid-point of the base located over T12. The base was secured also using double sided adhesive tape. This removed the requirement for an elastic thoracic strap. T12 was first located using the technique suggested in *Gray's Anatomy for Students* (Drake et al., 2008). This location was confirmed whilst the participant was in a flexed standing position, supporting themselves upon a stool, by palpation and counting of the spinous processes from this marked point down to the sacrum, and then double checked by counting back up to the marked spinous process.



Figure 12. Marker arrangement

3.4.1.4 Kinematic Data

Variability of angular kinematics of the lumbar spine about the three described axes relative to the pelvic segment was of primary interest (i.e. movement of the thoraco-lumbar marker cluster with respect to the pelvic markers). The Vicon Nexus software was used to run a Bodybuilder (Vicon, Oxford) code pipeline to calculate joint angles as outputs using Cardan (Euler) angles. The angles were calculated in the following order; 1) sagittal, 2) frontal, and 3) transverse. As with the biomechanical model, the Bodybuilder code used was the same as used by Schache et al. (2002^a; 2002^b). Data were filtered using a low pass Butterworth filter (fourth order, cutoff frequency determined for each individual participant as sum of residuals closest to zero using 4Hz, 6Hz, 8Hz, 10Hz, and

12Hz) and normalised to percentage gait cycle corresponding to initial right heel contact (0%) and subsequent right heel contact (100%). Heel contacts were identified as the lowest vertical displacement of a right heel marker. Stride duration and length was also calculated using the horizontal displacement of the right heel marker from initial right heel contact and subsequent right heel contact. Mean values for angular displacements, stride-to-stride intra-subject variability using CV_p and CV_o , were calculated for lumbar spine kinematics relative to the pelvis across all three planes of movement.

Intra-subject variability in the mean ensemble average has been typically calculated using Winter's (1983) coefficient of variation (Winter's CV) in studies of lumbar kinematic variability in chronic LBP (Vogt et al., 2001). Thus to ensure comparability between the population used in this study with the coefficient of variation reported in earlier study of chronic LBP participants, intra-subject variability was calculated using Winter's CV. However, the use of this method has recently been criticised due to the effect of waveform mean offsets altering relative variability away from the true variability in the system (O'Dwyer et al., 2009). O'Dwyer et al. (2009) noted that variability of mean offsets (CV_o) and waveform pattern variability (CV_p) should be calculated separately to account for the different information they provide; CV_o being determined by the reference frame used, identification of anatomical landmarks, markers and their configuration, whereas CV_p is more representative of repeatability of motor performance. Adding to this, the model used in this study has been examined for within-day repeatability previously and it was reported that marker reapplication errors and their effect upon daily mean offsets were the main source of concern (Schache et al., 2002^b). Thus Winter's CV (Eq. (1)), CV_p (Eq. (2)) and CV_o (Eq. (3)) were all calculated using the following equations:

$$CV = \frac{\sqrt{1/N \sum_{i=1}^N \sigma_i^2}}{1/N \sum_{i=1}^N |\bar{X}_i|} \times 100\%$$

Eq. (1)

Where N is the number of intervals over the stride period, X_i is the mean waveform at the i th interval and σ_i the standard deviations about X_i .

To counteract the effects of mean offset variability in examining the variability in the waveform pattern the raw waveforms for each subject may first be transformed to zero mean before averaging. The same approach as in Eq. (1) may then be employed to compute a CV for pattern variability (CV_p):

$$CV_p = \frac{\sqrt{1/N \sum_{i=1}^N (\sigma_i^{zero})^2}}{1/N \sum_{i=1}^N |\bar{X}_i^{zero}|} \times 100\%$$

Eq. (2)

Where $X_i(zero)$ is the average of the zero mean waveforms at the i th interval and $\sigma_i(zero)$ the standard deviation about $X_i(zero)$. $X_i(zero)$ is calculated:

$$\bar{X}_i^{zero} = \frac{1}{S} \sum_{j=1}^S X_{ji} - X_j^{offset}$$

Where S is the number of subjects, X_{ji} the raw waveform value for subject j at the i th interval and $X_j^{offset} = 1/N \sum_{i=1}^N X_{ji}$ the offset for subject j over all intervals in the stride period. $\sigma_i(zero)$ is calculated as:

$$\sigma_i^{zero} = \sqrt{\frac{\sum_{j=1}^S [(X_{ji} - X_j^{offset}) - \bar{X}_i^{zero}]^2}{S - 1}}$$

In line with the computation in Eq. (2), the standard deviation of the mean offsets of the raw data is also compared with the grand mean of the absolute value of the zero mean waveforms over all intervals:

$$CV_o = \frac{\sigma_o}{1/N \sum_{i=1}^N |\bar{X}_i^{zero}|} \times 100\%$$

Eq. (3)

Where $\sigma_o = \sqrt{\sum_{j=1}^S (X_j^{\text{offset}} - \bar{X}^{\text{offset}})^2 / (S - 1)}$ is the standard deviation of the pooled subjects' offsets and $\bar{X}^{\text{offset}} = (1/S) \sum_{j=1}^S X_j^{\text{offset}}$ the mean offset over all subjects.

This allowed differentiation of offset variability (CV_o) from pattern variability (CV_p), the latter being better representative of motor performance repeatability (for further discussion regarding the calculation please refer to O'Dwyer et al., 2009).

3.4.2 Participant Testing: Study 3

Participants underwent testing for ILEX strength and seated stadiometry at three points throughout the study (T1, T2, and T3). The ILEX test days were separated by at least 72 hours in order to avoid the effects of residual fatigue or soreness. At each time point participants were also required to complete the VAS and the ODI.

All stadiometry measurements were completed at the same time of day and participants were instructed to avoid heavy lifting for at least two days prior to testing (McGill et al., 1996). Measurements were conducted at the same time of day in order to control for diurnal variation. In order to normalise spine height prior to measurement the participant was instructed to lie in the supine position for 10 minutes with his or her hands resting on the stomach, head in a neutral position and supported by a pillow, and legs uncrossed with a pillow under the knees for support, as per the standard procedures used in the extant literature (Magnusson et al., 1996; Stothart & McGill, 2000; Rodacki et al., 2001; Kanlayanaphotporn et al., 2002; Rodacki et al., 2003). A custom set-up (See Figures 9 and 10) was used in combination with the same wall mounted stadiometer used for standing measurements. The wall mounted stadiometer calibration was checked before every test. Once 10 minutes elapsed participants were seated in the stadiometer setup

with their sacral crest against the rear board of the seat, hip, knee and ankle angles at 90°, and arms rested comfortably on a pillow across their lap. A line traced along the centre of the wooden seat was used to guide the participants in sitting centred when moving into the seat. The participants' feet were supported by mats to ensure hip, knee and ankle angles were at 90° with the number of mats used recorded and used during each test. Five anatomical points were identified and custom built adjustable rods were used to note the position of these for repeated testing (Healey et al., 2011). The points identified were: 1) the most posterior distension of the head; 2) the deepest point of the cervical lordosis; 3) the most prominent point of the thoracic kyphosis; 4) the deepest point of the lumbar lordosis; 5) the buttocks at the sacral crest (against the seat backboard). Control of these points (by noting during initial testing and replicating throughout further testing the vertical, horizontal and coronal position of the postural rods) ensured that participants adopted the same posture during all testing. After participants were seated in the stadiometer their heads were aligned in the Frankfurt plane to control their position and they were instructed to breathe in deeply maintaining their posture. They were instructed to hold their breath for 2-3 seconds whilst the head platform of the stadiometer was lowered until it made contact with the top of the head and measurement was taken. The testing was conducted by the present author. However, measurements were recorded by a research assistant and the results not disclosed to the primary investigator until both pre- and post-test data were collected in order to avoid investigator bias. The measurement dial on the stadiometer was obscured from the researchers' view during testing. Ten repeated measurements were taken over a period of ~3 - 3.5 minutes with the participant remaining in the stadiometer between measurements (Stothart & McGill, 2000). The reliability of this custom set-up was also examined and details of this investigation are provided in the appendices (Appendix 7.6).

3.5 Participant Training

In each of the studies the training intervention was as follows. Training was conducted at a frequency of 1x/week for a period of 12 weeks. This frequency of training has been shown to significantly improve ILEX strength and was chosen over more frequent training due to potential for overtraining when the lumbar extensor muscles are isolated (Graves et al. 1990^b). Also a second weekly training session offers no further improvements in symptomatic participants (Bruce-Low et al., 2012). Twelve weeks was the chosen duration as Carpenter et al (1991) have demonstrated that strength improvement from ILEX training occurs largely within the first 12 weeks. Participants performed one set of variable resistance ILEX exercise. In study 1 the FullROM group used their full ROM while the LimROM group only used the mid 50% of their individual ROM (Figure 13). In study 3 each participant trained using their full ROM. Resistance load was 80% of max recorded tested functional torque (TFT) during maximal isometric testing for both groups and repetitions performed until MMF in order to control for intensity of effort (Steele, 2013^b). Repetitions were performed taking at least 2 seconds to complete the concentric phase, holding for 1 second in full extension and taking at least 4 seconds for the eccentric phase. Resistance load was increased by 5% in the next session once the participant was able to continue exercise for over 105 seconds using his or her current load before achieving MMF.

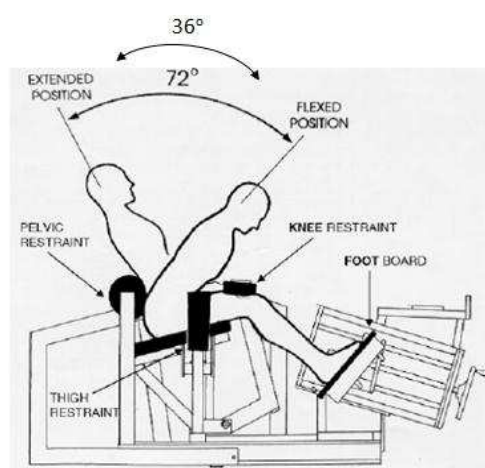


Figure 13. Example of limited ROM using the mid 50% of participants individual full ROM, in this case 72° (Adapted and reproduced with permission from MedX Corporation)

3.6 Data Analysis

Eligibility for analysis in each study required participants to have completed 75% (i.e. 9 out of 12 sessions) of the ILEX intervention within the 12 week intervention period. Isometric ILEX strength, recorded in units of torque, was measured across the participants' full ROM as foot pounds (ft.lbs^{-1}) and converted to Newton metres (Nm) using a correction of 1.356. Statistical analysis was performed using SPSS statistics computer package (vs.20) and $p \leq .05$ set as the limit for statistical significance. In addition, changes in pain and disability were also compared to consensus standards for MCIC (Ostelo et al., 2008). Ostelo et al., (2008) proposed the MCIC for VAS as 15mm and for ODI 10 points.

3.5.1 Data Analysis; Study 1

Twenty four participants (FullROM $n = 10$; LimROM $n = 7$; Control $n = 7$) data were available for analysis meeting the number required through power analysis. Because of individual differences between participants for lumbar ROM and subsequent determination of 50% ROM, ILEX strength data were further broken in quartiles (Q1 & Q4 corresponding to full extension and flexion respectively) for analysis with the fullROM group having trained throughout every quartile (Q1, Q2, Q3 & Q4) and the limROM group having only trained through the middle two quartiles (Q2 & Q3). Mauchly's test for sphericity was used to determine equality of variance between groups at $p > 0.05$. The independent variable examined was the training condition (i.e. FullROM, LimROM and Control) and dependent variables were absolute change in ILEX strength, lumbar ROM, standing ROM, pain and disability. Data with assumed sphericity for participant demographics and dependent variables were subjected to one way analysis of variance (ANOVA) to examine between group effects. Significant results from ANOVA were further subjected to Tukey post hoc tests to identify the location of differences. Pearson's correlations were also conducted between change in ILEX strength and change in VAS and ODI.

3.5.2 Data Analysis; Study 2

Twenty four participants' data (Males, $n = 13$; Females, $n = 11$) were available for analysis after allowing for attrition. Thus the number of participants combined with 5 trials per participant was sufficient for achieving adequate statistical power. Because of individual differences between participants for lumbar ROM, ILEX strength data were averaged across all angles tested. Mean values for angular displacements, stride-to-stride intra-subject variability using Winter's CV, CV_p and CV_o , were calculated for lumbar spine kinematics relative to the pelvis across all three planes of movement.

Demographic data met assumptions of normality and homogeneity of variance and thus were compared between groups at baseline using an independent samples t-test. Kinematic data did not meet assumptions of normality or homogeneity of variance as is typical for this type of data (Bates et al., 2004). Thus non-parametric statistical analysis was used and baseline kinematic data were compared between groups using the Mann Whitney-U exact test to check that randomisation had succeeded for these variables. For baseline kinematic variables (including means for displacements, stride-to-stride intra-subject variability using Winter's CV, CV_p and CV_o), Spearman's correlations were examined between them and VAS, ODI, and ILEX strength.

In examining the effects of the ILEX intervention the independent variable examined was participant group (i.e. Combined ILEX training or Control) and dependent variables were absolute change in the kinematic variables examined, VAS, ODI and ILEX strength. Wilcoxon Signed Ranks Exact test was used to compare across the independent conditions.

3.5.3 Data Analysis; Study 3

Nine participants' data (Males, $n = 4$; Females, $n = 5$) were available after allowing for attrition meeting the number required through power analysis. Because of individual

differences between participants for lumbar ROM, ILEX strength data were averaged across all angles tested. Mauchly's test for sphericity was used to determine equality of variance for data at $p>0.05$. The independent variable to examine was the time-point associated with the period (i.e. T1, T2, and T3) and dependent variables were the first measurement of each seated stature trial (Stad1st), average seated stature across the 10 measurements (StadAvg), shrinkage defined as the difference between the last and first of the 10 measurements (StadShrink; i.e. a negative value represented loss of seated stature), ILEX strength, lumbar ROM, pain and disability. Data with assumed sphericity for participant demographics and dependent variables were subjected to repeated measures ANOVA. Post hoc pairwise comparisons were conducted comparing T1 to T2 (encompassing the control period), T1 to T3 (encompassing both the control and intervention period) and T2 to T3 (encompassing the intervention period).

4. RESULTS & DISCUSSION

The following sections present the results from each of the three empirical studies in addition to their discussion.

4.1 A Randomised Controlled Trial of Limited Range of Motion Isolated Lumbar Extension Exercise in Chronic Low Back Pain Participants

4.1.1 Results

4.1.1.1 Participants

Participant baseline demographics are shown in table 6. ANOVA revealed no significant between group effects for age, stature, body mass, BMI, or symptom duration. Further, no significant between group effects were found for initial ILEX strength at any ROM quartile, or for lumbar ROM, standing ROM, pain, or disability. Attendance between training groups for ILEX training sessions did not significantly differ.

Table 6. Participant Baseline Demographics (Study 1)

	FullROM n = 10	LimROM n = 7	Control n = 7	<i>p</i>
Age (years)	46±12.36	41.86±17.45	41.7±15.1	0.838
Stature (cm)	173±8	174±0.08	180±8	0.510
Body Mass (Kg)	75.79±14.31	79±14.38	85.48±18.26	0.856
BMI	25.2±3.15	25.85±2.86	25.94±4.41	0.909
Symptom Duration (years)	12.99±12.03	14±10.86	11.85±10.59	0.859
ILEX Strength (Nm)				
Q1	116.34±35.48	153.03±84.60	141.3±52.67	0.431
Q2	158.11±66.74	197.69±76.79	194.11±58.97	0.505
Q3	189.09±89.34	235.75±102.39	212.96±55.79	0.491
Q4	231.67±91.52	265.58±108.72	279.06±83.70	0.760
Lumbar ROM (degrees)	64.5±12.1	68.57±6.8	62.7±6.24	0.497
Schobers Standing ROM (cm)				
SchFlex	21.66±1.57	21.66±1.00	21.31±1.32	0.549
SchExt	12.92±0.76	13.03±0.69	13.14±0.59	0.498
VAS (mm)	46.73±25.53	41.29±22.92	19.2±15.51	0.224
ODI (pts)	36.18±11.12	26.86±13.56	26.2±7.27	0.084
Attendance (%)	86.67±8.96	80.95±12.47	N/A	0.287
Gender Ratio (M:F)	5:5	3:4	5:2	N/A

Note: Results are mean ±SD

4.1.1.2 Isolated Lumbar Extension Strength

Figure 14 shows pre and post full ROM ILEX strength as strength curves plotted by quartile across the ROM. A significant between group effect was observed for change in ILEX strength at Q1 ($F_{(2, 21)} = 5.074$, $p = 0.016$, observed $\beta = 0.730$) Q2 ($F_{(2, 21)} = 5.976$, $p = 0.009$, observed $\beta = 0.878$), Q3 ($F_{(2, 21)} = 7.214$, $p = 0.004$, observed $\beta = 0.841$) and Q4 ($F_{(2, 21)} = 5.033$, $p = 0.016$, observed $\beta = 0.652$). Multiple comparisons using post hoc Tukey revealed no significant differences between FullROM and Lim ROM groups. FullROM increased significantly compared to Control at Q1 ($p=0.016$), Q2 ($p = 0.008$), Q3 ($p = 0.003$) and Q4 ($p = 0.024$). LimROM increased significantly when compared to Control at Q4 ($p = 0.034$). LimROM just failed to achieve significance when compared to Control at Q1 ($p = 0.059$), Q2 ($p = 0.060$) and Q3 ($p = 0.051$). Effect size for ILEX in FullROM and LimROM respectively were 2.08 and 1.80 at Q1, 1.74 and 1.29 at Q2, 4.74 and 3.45 at Q3, and 4.35 and 4.46 at Q4.

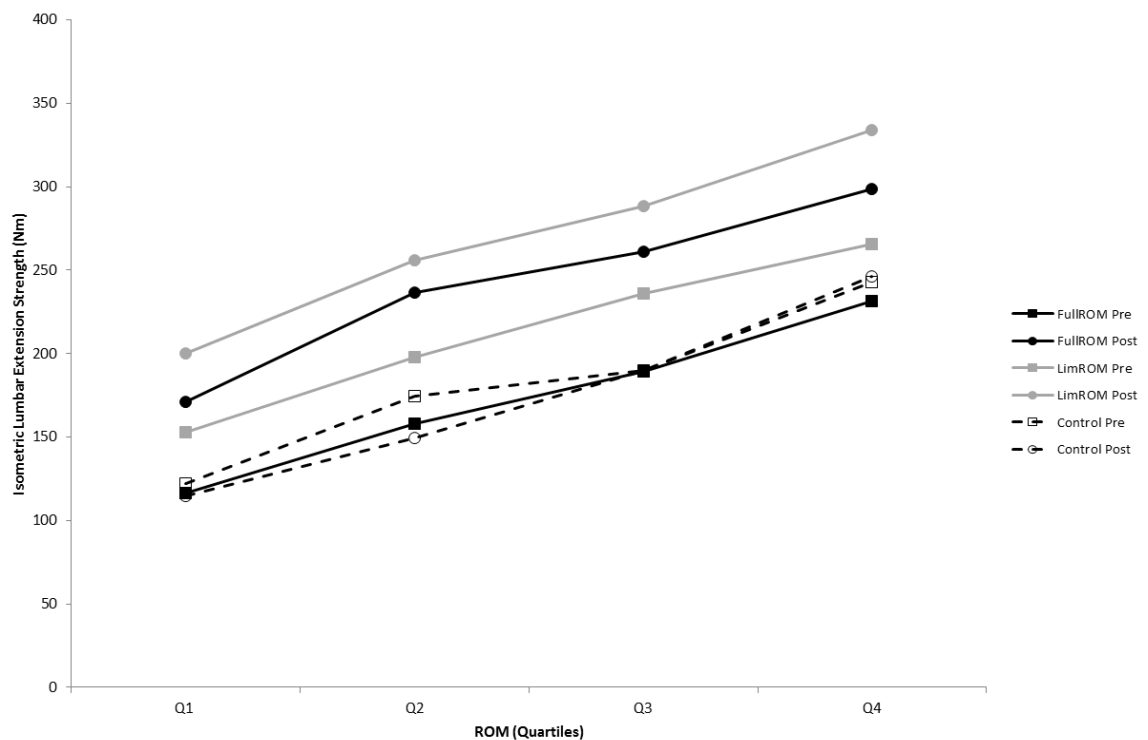


Figure 14. Pre and post mean isometric ILEX strength curves plotted by quartile across the ROM.

4.1.1.3 Lumbar and Standing Range of Motion

Absolute changes in both lumbar and standing ROM are shown in table 7. ANOVA revealed no significant between group effects were seen for lumbar ROM ($F_{(2, 21)} = 2.882$, $p = 0.067$, observed $\beta = 0.504$), SchFlex ($F_{(2, 21)} = 0.157$, $p = 0.856$, observed $\beta = 0.071$), and SchExt ($F_{(2, 21)} = 0.644$, $p = 0.536$, observed $\beta = 0.142$).

Table 7. Change in lumbar and standing ROM.

	FullROM	LimROM	Control
Lumbar ROM (degrees)	5.1±6.01	0.43±1.13	1.8±2.09
Schobers Standing ROM (cm)			
SchFlex	0.18±1.29	0.05±0.94	-0.22±0.62
SchExt	-0.15±0.65	0.17±0.49	-0.05±0.32

Note: Results are mean ±SD

4.1.1.4 Pain and Disability

Absolute changes in VAS and ODI are shown in table 8. A significant between group effect was observed for change in VAS ($F_{(2, 21)} = 8.263$, $p = 0.002$, observed $\beta = 0.966$) and ODI ($F_{(2, 21)} = 12.586$, $p < 0.001$, observed $\beta = 0.998$). Multiple comparisons using post hoc Tukey revealed no significant differences between FullROM and LimROM groups for either change in VAS or ODI. FullROM VAS scores decreased significantly compared with control ($p = 0.002$). Change in VAS for LimROM approached significance when compared with control ($p = 0.058$). FullROM and LimROM both decreased scores for ODI significantly compared with control ($p < 0.001$ and $p = 0.023$ respectively). Effect size for VAS in FullROM and LimROM respectively were 1.67 and 0.9. Effect size for ODI in FullROM and LimROM respectively were 2.28 and 1.5. Changes in VAS achieved the MCIC for FullROM (-30.3±25.76mm) and LimROM (-16.29±10.97mm) groups. MCIC was also achieved for changes in ODI for FullROM (-18.2±6.63pts) and LimROM (-12±5.16pts). The control group did not achieve MCIC values for either or VAS (10.29±18.12mm) or ODI (-1.71±7.95pts).

Pearson's correlation revealed significant moderate correlations between change in ILEX strength and VAS for Q1 ($r = -.484$, $p = 0.017$), Q2 ($r = -.595$, $p = 0.002$), Q3 ($r = -.651$, $p = 0.001$), and Q4 ($r = -.464$, $p = 0.022$) indicating that a greater increase in ILEX strength was associated with a greater decrease in VAS score. No significant correlations were seen between change in ILEX strength and change in ODI for Q1 ($r = -.261$, $p = 0.219$) or Q4 ($r = -.390$, $p = 0.060$). Significant moderate correlations were shown for Q2 ($r = -.453$, $p = 0.026$), and Q3, ($r = -.522$, $p = 0.009$).

Table 8. Change in VAS and ODI (Study 1)

	FullROM	LimROM	Control
VAS (mm)*	-30.3±25.76 [†]	-16.29±10.97 [†]	10.29±18.12
ODI (pts)*	-18.2±6.63 [†]	-12±5.16 [†]	-1.71±7.95

Note: Results are mean±SD; * $p \leq 0.05$ between groups comparison with ANOVA; † $p \leq 0.05$ between training group (FullROM/LimROM) and Control

4.1.2 Discussion

This study is first to demonstrate comparable effects of both full ROM and limited ROM ILEX exercise in chronic LBP participants upon ILEX strength, pain and disability. Absence of statistically significant differences between the FullROM and LimROM training groups for increases in ILEX strength (~22% to ~54%) at all parts of the ROM suggest that limited ROM exercise does increase strength throughout the full ROM, including untrained areas, thus confirming the original hypothesis. These increases are also comparable to those obtained through full ROM exercise. These results in chronic LBP participants are in accordance with those seen in asymptomatic participants for ILEX (Graves et al., 1992^b) as well as other exercises (Knapik et al., 1983; Graves et al., 1989; Massey et al., 2004). The increases in ILEX strength for the FullROM group compared with the control group were significant and comparable to other research examining symptomatic chronic LBP participants (Risch et al., 1993; Mooney et al., 1993; Nelson et al., 1995; Mooney et al., 1995; Holmes et al., 1996; Leggett et al., 1999; Nelson et al., 1999; Choi et al., 2005; Smith et al., 2011; Bruce-Low et al., 2012). Though the LimROM

group was significantly greater than the control group at Q4, that they did not attain significance at other Qs is likely the result of a type II error (i.e. failure to reject the null hypothesis) as changes were comparable and did not significantly differ from FullROM, and that ESs were both large and similar between both training groups.

Lumbar ROM measured using the MedX Lumbar Extension Machine and standing ROM measured via the modified schobers method showed no significant changes as a result of the intervention. The lack of a treatment effect upon lumbar ROM is similar to some studies that showed no significant improvement in ROM as a result of ILEX training (Leggett et al., 1999; Smith et al., 2011) yet contrasting to others that have demonstrated significant improvement (Nelson et al., 1995; Bruce-Low et al., 2012). A possible reason for the lack of difference in lumbar ROM in this study however may be the sample used. Baseline lumbar ROM in studies showing an improvement (54° to 65°; Nelson et al., 1995; Bruce-Low et al., 2012) compared with the baseline lumbar ROM of participants in this study (65° to 68°) and others that have not shown an improvement (64°; Smith et al., 2011) is generally lower, though others do not follow this tendency (55° to 62°; Leggett et al., 1999). A lower beginning lumbar ROM might offer greater potential for lumbar ROM increases. There is scope for future research to examine this through identification and recruitment of those who already have a substantially limited lumbar ROM.

No significant differences as a result of the intervention were shown for any of the schobers methods i.e. SchFlex, or SchExt. In contrast other research has shown improvements in SchFlex as a result of training using ILEX in chronic LBP participants (Bruce-Low et al., 2012). The reasons for this discrepancy may be similar to the reasons for the lack of difference in lumbar ROM measured using the MedX Lumbar Extension Machine. Participants in the study by Bruce-Low et al., (2012) had a baseline SchFlex ranging from ~14cm to 19cm whereas participants in this present study had greater baseline measurements of ~21cm. Again there may have been less potential for an

improvement. For example, the baseline SchFlex values of our participants were similar to the asymptomatic normative data (~21cm) presented by MaCrae & Wright (1969) and SchExt values of our participants were also similar to the asymptomatic participants (~13.5cm) in the study by Beattie et al. (1987) which significantly differed from their asymptomatic population (~14.5cm). Sagittal ROM is limited in association with severity of degenerative changes, particularly the disc (Mimura et al., 1994), in order to maintain stability. Thus perhaps our participant sample were not severely degenerative. An alternative explanation may be due to the inherent sources of error in tests such as the schobers. Although a widely used method (Schober, 1937; MaCrae & Wright, 1969), unavoidable sources of error exist, the most prominent being palpation and identification of anatomical landmarks (Loebl, 1967; MaCrae & Wright, 1969; Mootz et al., 1989; Harlick et al., 2007). Lumbar ROM measured using the MedX Lumbar Extension Machine is instead a more valid technique (Shirley et al., 1994).

In this present study, and in line with our hypothesis, both FullROM and LimROM demonstrated significant reductions in pain and disability as measured by VAS and ODI compared with the control group. No significant difference was observed between the two training groups. Previous research demonstrated that ILEX produces significant and meaningful reductions in both pain and disability in chronic LBP participants (Risch et al., 1993; Mooney et al., 1993; Nelson et al., 1995; Mooney et al., 1995; Deutsch, 1996; Holmes et al., 1996; Nelson et al., 1999; Leggett et al., 1999; Choi et al., 2005; Smith et al., 2011; Bruce-Low et al., 2012). These studies, however, utilised full ROM exercise in all instances. The results of the present study confirm that the use of full ROM ILEX exercise is not necessary for significant and meaningful improvement of pain or disability.

Recently, international consensus has been offered on the MCIC for pain and disability (Ostelo et al., 2008). The changes demonstrated in VAS and ODI for both FullROM (-30.3±25.76mm and -18.2±6.63points respectively) and LimROM (-16.29±10.97mm and -

12±5.16points respectively) achieved the MCICs as set out in this consensus whereas the control group did not (10.29±18.12mm and -1.71±7.95points respectively). As such it would seem that the results of this study demonstrate both full and limited ROM ILEX exercise produce comparable improvements in ILEX strength, at all parts of the ROM, in addition to significant and meaningful improvements in both pain and disability in chronic LBP participants

It has recently been questioned whether reductions in pain and disability are in any way attributable to corresponding improvements in muscular function (Steiger et al., 2012^a). However, research using ILEX was not considered in this review (Steele & Bruce-Low, 2012; Steiger et al., 2012^b). It may be that the exercises used in the studies considered were not specific enough to condition the lumbar extensors. Further, an issue raised was that many have not reported correlations between these outcomes (Steiger et al., 2012^a). As such we considered this within the present study demonstrating significant moderate correlations between change in ILEX strength and pain ($r = -.488$ to $-.668$), as well as change in ILEX strength and disability ($r = -.414$ to $-.539$) indicating the greater the improvement in ILEX strength the greater the reduction in pain and disability. In contrast with the lack of correlations between change in pain and disability with muscular performance in the studies examined by Steiger et al., (2012^a), our findings may be due to the specific nature of the ILEX exercise, or that lumbar extensor muscular function was measured in isolation. However, this requires further clarification, as from this data it cannot be determined specifically whether increases in strength affected decreases in pain or vice versa.

It appears evident from this study that limited ROM ILEX exercise is as effective as full ROM ILEX exercise in for improving ILEX strength throughout a participants' full ROM in addition to reducing pain and disability. Thus, recommendations to prescribe limited ROM exercise for those with chronic LBP (Graves et al. 1992^b; McGill, 2007) now have a basis

in empirical evidence. There is also potential practical benefit of these recommendations considering other findings regarding ROM and pain. Movement to full extension or flexion has impacts upon pain experienced in symptomatic participants (Donelson et al., 1991). In addition, functional status and pain improves to a significantly greater degree with exercise focused upon participants' directional preference (i.e. avoiding flexion or extension based upon individual preference; Werneke et al., 2011). Avoidance of painful positions during exercise also impacts upon adherence in chronic LBP participants. Long et al. (2004) reported that one third (~33%) of oppositely matched and non-directional preference participants withdrew during their study whereas no dropouts were observed in the group that were directionally matched. This may have been due to participants matched to directional preference experiencing less pain during exercise. No differences were apparent in dropout rates for either of the groups in this present study and no significant differences were found for attendance or improvements. Long et al. (2004) only performed a 2 week intervention common to the type of intervention they used. The present study was 12 weeks in length and showed lower participant attrition once participants had begun the training (average ~14%, see Figure 6). Perhaps with a longer intervention period any differences may become apparent between the two groups (FullROM and LimROM). Alternatively the results could indicate that ILEX is an approach that maintains participant adherence. Again this is a worthwhile avenue for future research.

A last point may be made regarding the safety of exercise as a form of treatment for chronic LBP. Though effective, active treatment in the form of exercise may hold potential for re-injury. In this respect limited ROM ILEX exercise may be safer for chronic LBP participants. It has been shown that cumulative fatigue (such as that experienced during a set of fatiguing exercise) produces greater spinal flexing (Van Dieen et al., 1998; Dolan & Adams, 1998; Sparto et al., 1997^a; Sparto et al., 1997^b) which nears the proposed margin of safety for intervertebral disc mechanical injury (Dolan & Adams, 1998). Limiting the

ROM during exercise may combat this more effectively than full ROM exercise. However, the present study was not of sufficient design and size to determine this and, as with other points made in this discussion, this is an area requiring further research.

Chronic LBP is a multifactorial condition with a wide variety of dysfunctions including lumbar extensor deconditioning and limited sagittal ROM. As a result, limited ROM exercise is often prescribed as treatment and the aim of this study was to examine this recommendation. The results of this study demonstrated both full ROM and limited ROM ILEX exercise improve ILEX strength throughout a full ROM in addition to reducing pain and disability. From a clinical perspective these improvements achieve MCIC for perceived pain and disability thus both approaches could be deemed appropriate for chronic LBP participants.

4.2 Lumbar Kinematic Pattern Variability during Gait and the Effects of Isolated Lumbar Extension Exercise in Chronic Low Back Pain Participants

4.2.1 Results

Results for ILEX strength, VAS, ODI standing ROM and lumbar ROM were already presented in this report. Thus, to avoid replication, only kinematic data and relationships examined between VAS, ODI, ILEX strength and kinematic variables are reported here.

4.2.1.1 Participants

Participant demographics, pain, disability and ILEX strength data are shown in Table 9 for groups. Comparison between groups revealed that most demographic variables at baseline did not significantly differ thus it was considered that randomisation had been successful. The only significantly different characteristic between groups was VAS score ($t_{(22)} = 2.420$, $p = 0.024$).

Table 9. Participant group demographics (Study 2)

	Training (n = 17)	Control (n = 7)	<i>p</i>
Age (years)	47 \pm 13	44 \pm 16	0.645
Stature (cm)	171.90 \pm 9.26	180.02 \pm 8.92	0.076
Body Mass (Kg)	75.00 \pm 15.49	82.92 \pm 19.37	0.324
BMI (Kg/m ²)	25.12 \pm 3.10	25.33 \pm 4.36	0.899
Symptom Duration (years)	14 \pm 11	12 \pm 10	0.800
VAS (mm)	47.26 \pm 24.09	23.00 \pm 16.62	0.024
ODI (pts)	34.71 \pm 12.69	27.15 \pm 7.65	0.158
Lumbar Extension Strength (Nm)	177.80 \pm 83.80	192.21 \pm 67.60	0.691
Gender Ratio (M:F)	8:9	5:2	

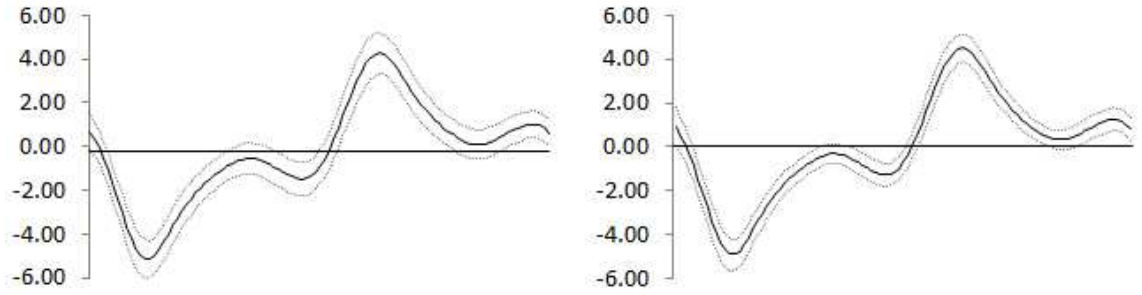
Note: Results are mean \pm SD

4.2.1.2 Baseline Kinematic Data

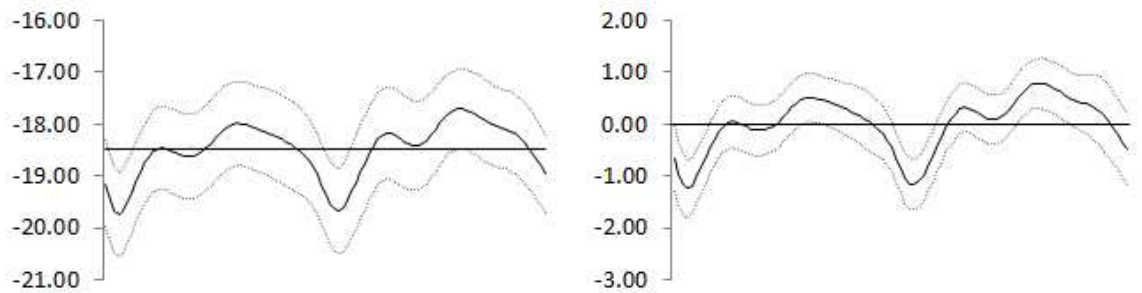
Between group comparisons again revealed that the majority of kinematic variables did not significantly differ at baseline, only sagittal CV_o ($U = 23.000$, $Z = -2.318$, $p = 0.019$), and both transverse Winter's CV and CV_o (respectively; $U = 17.000$, $Z = -2.699$, $p = 0.005$).

Due to inclusion of novel methods of determining ensemble average variation in this study (CV_p and CV_o ; O'Dwyer et al., 2009), compared with others use of Winter's CV research (Vogt et al., 2001), baseline data were pooled for all participants in order to compare Winter's CV, CV_p , and CV_o in this population of chronic LBP participants. Displacement and Winter's CV were highest and similar in frontal and transverse planes. Contrastingly CV_p and CV_o were higher in the sagittal plane than in frontal and transverse planes which were both also similar. Figure 15 presents a comparison of these pooled data showing mean and SDs with Winter's CV, and mean and SDs transformed to zero with both CV_p and CV_o . It can be noted that, particularly for the sagittal plane, Winters CV does not offer an accurate reflection of the variability across the ensembled waveform patterns (SD reflected by width of dotted lines) whereas CV_p does.

[a] Frontal – Winter's CV 34.74 % - CV_p 45.07% – CV_o 28.88%



[b] Sagittal – Winter's CV 6.73% - CV_p 106.44% - CV_o 92.97%



[c] Transverse – Winter's CV 38.66% - CV_p 42.81% - CV_o 33.66%

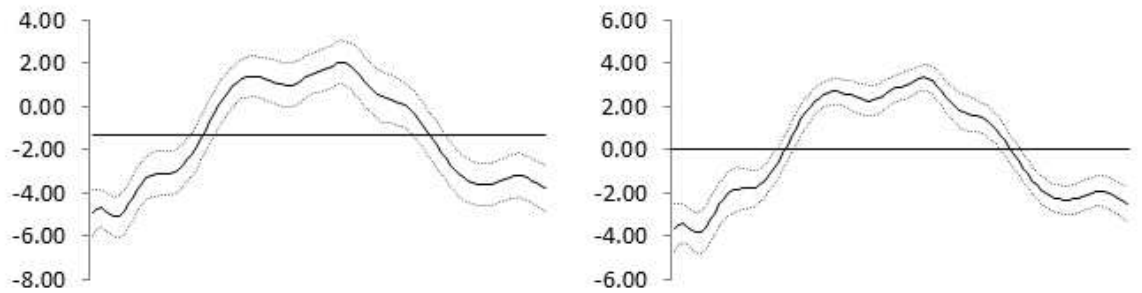


Figure 15. Waveform patterns, Winter's CV, CV_p and CV_o. The x axis of each graph reflects the gait cycle (0-100%) with the y axis reflecting angular displacement (movement amplitude in degrees). Waveform patterns, Winters CV, CV_p and CV_o. Left hand graphs from top to bottom show mean (\pm standard deviation) for [a] frontal, [b] sagittal and [c] transverse lumbar spine kinematics and Winter's CV respectively, with offsets shown by the horizontal axis, for pooled data ($n = 24$). Right hand graphs from top to bottom show mean (\pm standard deviation) with raw signals transformed to zero mean removing offsets and CV_p and CV_o for the same data.

Spearman's correlations revealed a significant moderate positive correlation between VAS and only sagittal plane Winter's CV ($r = .411$, $p = 0.023$). Significant moderate positive correlations were found between ODI and sagittal plane Winter's CV ($r = .457$, $p = 0.012$), transverse plane Winter's CV ($r = .404$, $p = 0.025$) and transverse plane CV_p ($r = .401$, p

0.026). Significant moderate negative correlations were also found between ILEX strength and frontal plane CV_o ($r = -.370$, $p = 0.045$), sagittal plane Winter's CV ($r = -.467$, $p = 0.014$), transverse plane Winter's CV ($r = -.435$, $p = 0.021$), transverse plane CV_p ($r = -.411$, $p = 0.029$), transverse plane CV_o ($r = -.378$, $p = 0.042$) and a significant moderate positive correlation with transverse plane displacement ($r = .442$, $p = 0.020$).

4.2.1.3 Effects of Intervention upon Kinematic Variables

Table 10 shows pre and post data for displacement, Winter's CV, CV_p and CV_o . Figure 16 presents an example participant's data to demonstrate change in the waveform pattern variability. Wilcoxon Signed Ranks Exact test revealed significant changes from pre to post only for sagittal plane CV_p ($W_{(16)}$, $Z = -1.728$, $p = 0.044$) in the training group only suggesting improvement in stride to stride waveform pattern replication after the intervention. Effect size for sagittal CV_p in the training group was 0.48.

Table 10. Pre and Post Kinematic data

	Displacement (degrees)			CV _p (%)			CV _o (%)		
	Frontal	Sagittal	Transverse	Frontal	Sagittal	Transverse	Frontal	Sagittal	Transverse
Training									
Pre	10.61±3.74	3.92±1.20	8.85±2.72	41.95±16.62	111.99±42.64	46.49±20.57	27.48±18.34	103.94±52.78	41.69±28.15
Post	10.80±2.88	4.31±1.37	9.41±3.26	39.35±12.72	91.09±28.27*	48.20±24.02	25.87±15.02	87.95±41.10	42.35±25.28
Control									
Pre	8.15±1.94	4.13±1.78	6.91±7.87	52.65±19.23	92.95±27.07	33.41±11.74	32.30±29.09	66.33±69.07	14.15±5.46
Post	7.25±2.31	3.80±1.54	8.86±2.32	56.45±11.82	89.51±26.63	40.25±20.83	44.59±46.13	85.91±39.78	31.66±27.27

* Denotes significant changes from pre to post

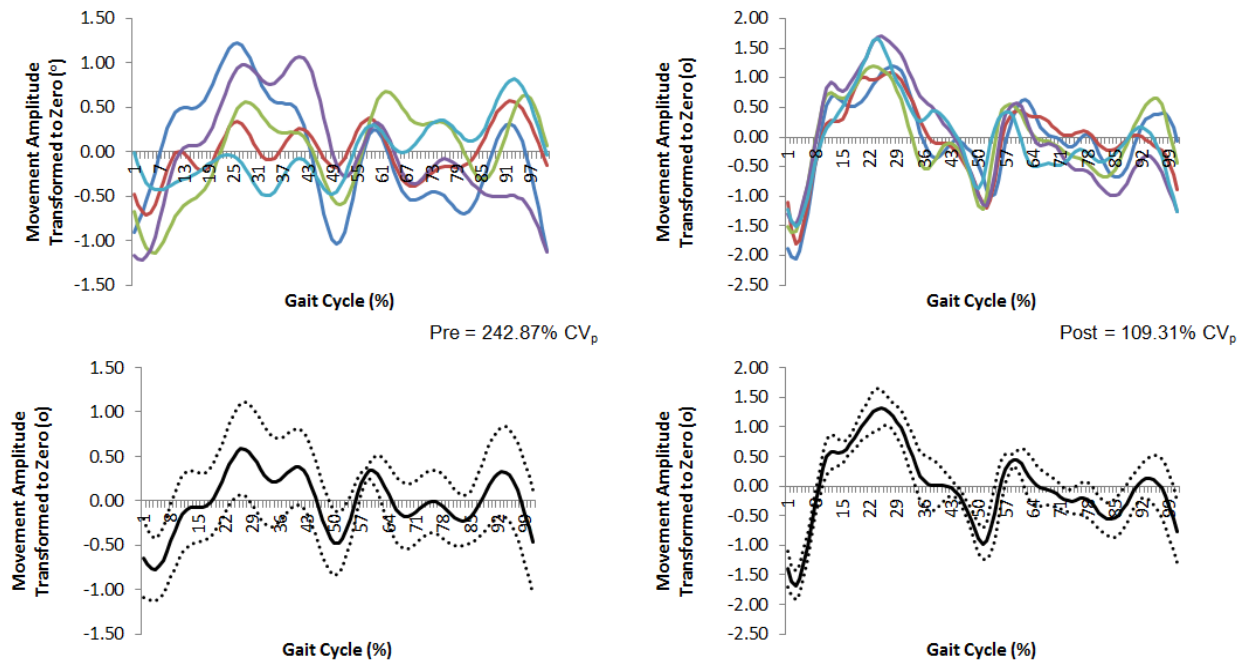


Figure 16. Example participant data showing pre and post individual trial waveforms and pre and post waveform variability transformed to zero (CV_p)

4.2.2 Discussion

This study yields several novel findings: 1) sagittal plane lumbar kinematic waveform patterns appear to be considerably more variable than frontal or transverse planes in chronic LBP, observed using novel methods of differentiating offset variability from pattern variability in this population and in contrast to earlier studies using Winter's CV, 2) transverse plane lumbar pattern variability is significantly correlated with ILEX strength and ODI, and 3) a 12 week ILEX resistance training intervention significantly improves sagittal plane pattern variability during gait in chronic LBP participants. These findings potentially offer further understanding regarding relationships between chronic LBP, gait variability and lumbar extensor deconditioning.

Within this study the foremost interest was repeatability of lumbar movement patterns exhibited (intra-subject stride-to-stride variability) as, despite similar average movements occurring amplitudes, symptomatic participants appear less able to replicate these consistently (Vogt et al., 2001). Vogt et al. (2001) reported data using Winter's CV suggesting lumbar movement variability during gait was significantly higher in chronic LBP participants compared with asymptomatic controls, and that sagittal and transverse plane variability was greater than frontal plane variability. For comparison with previous research Winter's CV was calculated for the present study's data. Results for Winter's CV differed from those of Vogt et al. (2001) where sagittal plane variability appeared lowest (Vogt et al. 2001 – 26.93%; Present study – 6.73%), and both frontal and transverse plane variability was slightly higher (Vogt et al., 2001 – 14.87% and 26.45% frontal/transverse respectively; Present study – 34.74% and 38.66% frontal/transverse respectively).

This difference in sagittal plane Winter's CV might be accounted for by the large mean offset in the data's waveform. Vogt et al. (2001) calibrated their measurements to angles during the standing posture to zero their measurements whereas in the present study they were not. The sagittal plane data were instead similar to that reported by Lamothe et al.

(2002). A large mean offset effectively deflates Winter's CV (O'Dwyer et al., 2009). Because of this O'Dwyer et al. (2009) suggested differentiating the offset from calculation of the variability in the waveform pattern; the latter they suggest being more representative of movement replication whereas the offset incorporates a greater variation from other sources (i.e. marker error). Indeed Schache et al. (2002^b) reported that, although the model used here displayed high within-day repeatability, angular parameters were most susceptible to marker reapplication errors from repeated measures affecting waveform offset.

The data show CV_p differs considerably from variation calculated using Winter's CV. Sagittal plane variation (106.44%) is more than double that in frontal (45.07%) and transverse planes (42.81%). Figure 15 shows that CV_p better represents the absolute variation in the waveform (the standard deviations depicted by the dotted lines) as noted by sagittal plane standard deviation bandwidth being twice the width of frontal and transverse planes. Winter's CV on the other hand does not represent this in the raw data as it is clear both frontal and transverse plane variance are not ~5 times larger than sagittal plane variance. This further demonstrates that differentiation of offset and pattern variability better represents motor performance repeatability and is less affected by inter-individual marker application errors affecting mean offset values for individual participants.

CV_p has not yet been calculated in chronic LBP participants, thus it is not possible to verify whether greater sagittal plane pattern variability is a typical characteristic of their gait. Nor is it possible to define the clinical meaning of this in comparison to healthy gait as CV_p has not been reported for lumbar spine gait kinematics in asymptomatic participants to the author's knowledge. The correlation data suggest that those with lower ILEX strength exhibit higher sagittal and transverse plane variability when considering Winter's CV. However, the inherent limitation of this method described above must be taken into account. Yet, despite high sagittal plane CV_p in comparison to other planes, baseline

correlation results suggest there is instead a relationship between ILEX strength and transverse plane kinematics; lower transverse displacement and higher CV_p being associated with lower ILEX strength. This relationship may be a consequence of the lumbar extensor deconditioning associated with LBP. Indeed extensor fatigue impacts lumbar kinematics during gait emphasising the link between deconditioning and gait abnormality (Harts et al., 2009).

Lumbar control during healthy gait is undoubtedly aided by the musculature. Most studies examining muscular contributions to gait have identified the active role the lumbar extensor musculature plays (Thorstensson et al., 1982; Winter et al., 1993; Callaghan et al., 1999). Thorstensson and colleagues (1982) showed the pattern of the multifidus and longissimus activation during gait involved two bursts of activity per cycle each corresponding to foot strike. They concluded this activity, in relation to trunk movement pattern, suggested the lumbar extensors' main function during gait is to control excessive trunk movement. Callaghan and colleagues (1999) demonstrated similar bimodal activity corresponding to greater peak in the musculature ipsilateral to the contacting foot. Lumbar extensor activity appears to follow a pattern to stabilise superior segments against inertial and gravitational forces during both single foot contacts (Thorstensson et al., 1982; Winter et al., 1993; Callaghan et al., 1999).

It seems reasonable that in chronic LBP, wherein there is associated deconditioning of what appears to be critically important musculature for controlling gait, that the deconditioning of this musculature might be responsible for gait abnormality. Indeed these results tend to support this with respect to transverse plane CV_p during gait. Some have reported that transverse plane kinematics typically show *lower* variability in those with chronic LBP (Lamoth et al., 2002; Lamoth et al., 2006^a; van der Hoorn et al., 2012). However, these studies have examined trunk and pelvis *coordination* and variability in phase differences whereas the present study examined lumbar waveforms relative to the

pelvis. This difference in methods may explain the different conclusions between studies. The present study also suggests low ILEX is associated with smaller transverse displacements. Perhaps transverse movement is more rigid in chronic LBP, yet within that smaller range of movement there is poor waveform pattern repeatability. The rigidity seen in transverse kinematic coordination in chronic LBP (Lamoth et al., 2002; Lamoth et al., 2006^a; van der Hoorn et al., 2012) may still be a manifestation of lumbar extensor deconditioning. Considering this future research should examine the relationship between ILEX and trunk/pelvis coordination in those with chronic LBP.

These results also provide further corroborating evidence against the idea that pain *per se* (i.e. its presence as an independent factor) causes abnormal gait in chronic LBP. Indeed it has been found that neither induced pain, fear or pain, or history of pain once resolved affect muscle activity independently (Lamoth et al., 2004; Seay et al., 2011^a; 2011^b). Although significant positive correlation was found between VAS and Winter's CV, no significant correlation was found between VAS and CV_p or any other kinematic variable supporting others findings that pain presence is not associated with gait variability (Lamoth et al., 2004; Anders et al., 2005; Seay et al., 2011^a). There was, however, a significant correlation between ODI and transverse plane CV_p. Considering LBP's multifactorial nature this suggests gait variability is potentially a symptom associated with chronic LBP that results from lumbar extensor deconditioning or the disability accompanying chronic LBP.

Regarding baseline observations, a limitation of the present study was the lack of a comparable healthy control group due to the study's initial design as an experimental trial. Winter's CV data suggest that chronic LBP participants show higher lumbar spine variability compared to data from normal participants in earlier studies (Vogt et al., 1999; Vogt et al., 2001). Thus the variability identified from CV_p data would likely be greater in the chronic LBP participants in this study compared with healthy controls. However, CV_p

has not been calculated for lumbar spine kinematics in healthy participants in the published literature. Thus future work in healthy participants should utilise this method (O'Dwyer et al., 2009) to produce normative data for comparison and to judge improvement from clinical intervention.

The baseline association between weak ILEX strength and greater variability, however, supports the notion that exercise might be a valuable intervention. Indeed, previous studies have supported exercise based interventions for improving aspects of gait including muscle activation (Tsao & Hodges, 2008), ground reaction force parameters (Da Fonseca et al., 2009) and displacements during gait (Carpes et al., 2008). However, none have examined lumbar kinematic variability during gait, nor utilised specific exercise designed to isolate the lumbar extensors. The results indicate that ILEX resistance training significantly reduced sagittal plane CV_p, suggesting greater ability of participants to replicate motor patterns in this plane during gait.

Baseline data indicated a relationship between transverse CV_p and ILEX strength yet the ILEX intervention reduced sagittal CV_p. Unlike previous research findings that Winter's CV was low in chronic LBP participants (Vogt et al., 2001), sagittal plane CV_p was found to be highest in this population and so may explain improvements observed as there may have been the greatest scope for improvement. However, the significant improvement ($-20.90 \pm 43.53\%$) in sagittal CV_p may suggest a specific intervention effect due to the plane of motion that ILEX exercise is performed through. An exercise device similar to the one used in this study for ILEX also exists that allows pelvic restraint for torso rotation through the transverse plane to be performed in isolation (Torso Rotation Machine, MedX Corporation, Ocala, Florida). Mooney et al. (2001), after demonstrating that the latissimus dorsi and contralateral gluteus maximus are reciprocally active during gait, presumably contributing to transverse plane control, further examined the effects of isolated torso rotation exercise. In this study they reported abnormal activation patterns in symptomatic

participants compared with controls during torso rotation exercise. After an intervention of progressive resistance training using the torso rotation device this activation returned to normal patterns seen in asymptomatic controls. However, despite reporting EMG results for the latissimus and gluteus to clarify their role during gait, Mooney et al. (2001) did not perform pre and post intervention measurements to identify if any change had occurred in muscular control during gait. In light of the present studies results, future research should quantify whether plane of movement specific training produces consequent plane of movement specific changes in control of the lumbar spine during gait e.g. whether torso rotation improves transverse plane CV_p .

This study has provided novel information on lumbar spine kinematic variability during gait in chronic LBP through use of novel methods of analysing pattern variability. These findings contrast with those utilising Winter's CV and instead suggest the highest variability occurs in sagittal plane lumbar movement during gait. Further, there was a significant relationship between both ILEX strength and ODI with transverse plane lumbar CV_p , and a lack of relationship between VAS and CV_p in any plane. The 12 week ILEX resistance exercise significantly improved sagittal plane CV_p indicating improved motor pattern replication. These findings demonstrate that improvements are possible in various dysfunctions typically associated with chronic LBP through use of ILEX exercise and add to understanding of the multifactorial relationships between them and lumbar extensor deconditioning in LBP.

4.3 The Effects of Isolated Lumbar Extension Exercise Upon Disc Hydration Measured Indirectly through Seated Stadiometry in Participants with Chronic Low Back Pain

4.3.1 Results

4.3.1.1 Participants

Participant baseline demographics are shown in table 11.

Table 11. Participant Baseline Demographics (Study 3)

	Participants (n = 9)
Age (years)	51±12
Stature (cm)	167.7±6.9
Body Mass (Kg)	77.46±13.94
BMI	27.4±3.2
Symptom Duration (years)	15±14
ILEX Strength (Nm)	195.42±109.99
Lumbar ROM (degrees)	65.7±10.1
VAS (mm)	33.4±23.3
ODI (pts)	26.7±11.2

Note: Results are mean ±SD

5.3.1.2 Seated Stadiometry

Table 12 shows results from seated stadiometry testing at each time point. ANOVA revealed no significant repeated measures effects were found for Stad1st ($F_{(2, 16)} = 0.404$, $p = 0.674$, observed $\beta = 0.104$), StadAvg ($F_{(2, 16)} = 0.422$, $p = 0.663$, observed $\beta = 0.107$) or StadShrink ($F_{(2, 16)} = 0.636$, $p = 0.542$, observed $\beta = 0.138$) respectively.

Table 12. Seated stadiometry result from each time point (T1, T2, and T3).

	T1	T2	T3
Stad1st (mm)	864.2±33.5	866.2±37.4	867.1±38.1
StadAvg (mm)	863.6±34.7	862.5±37.0	864.6±39.1
StadShrink (mm)	-1.3±3.3	-5.0±7.3	-3.1±6.3

Note: Results are mean ±SD

4.3.3 Isolated Lumbar Extension Strength

Figure 17 shows ILEX strength measured at each time point. A significant repeated measures effect by time was observed for ILEX strength ($F_{(2, 16)} = 26.263$, $p < 0.0001$, observed $\beta = 1.000$). Post hoc pairwise comparisons revealed a significant difference between both T1 and T3 ($p = 0.002$) and T2 and T3 ($p < 0.0001$). Effect size for change in ILEX between T2 and T3 was 2.20.

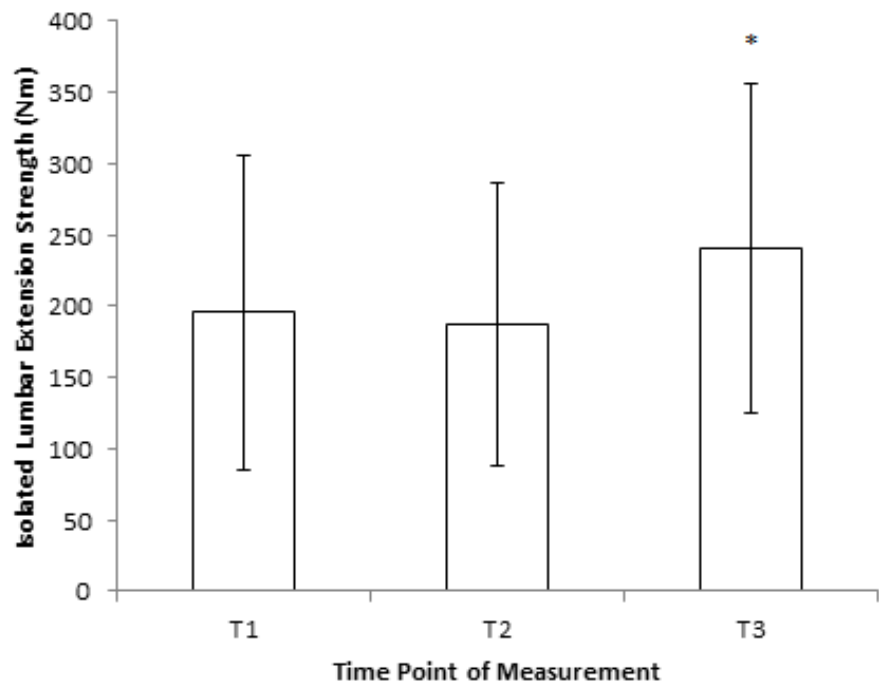


Figure 17. ILEX strength at each time point. Note; *indicates significant pairwise comparison between both T1 and T3, and T2 with T3

4.3.1.4 Pain and Disability

VAS and ODI measures for each time point are shown in table 13. ANOVA failed to achieve significance for repeated measures effect by time for VAS ($F_{(2, 16)} = 3.281$, $p = 0.064$, observed $\beta = 0.539$). A significant repeated measures effect by time was observed for ODI ($F_{(2, 16)} = 6.846$, $p = 0.007$, observed $\beta = 0.862$). Post hoc pairwise comparisons revealed a significant difference between T1 and T3 ($p = 0.037$) for ODI. Effect size for change in VAS and ODI respectively between T2 and T3 was 0.98 and 1.42. Changes in VAS and ODI over the control period (between T1 and T2) did not achieve MCICs.

Changes in VAS and ODI after the intervention period (between T2 and T3) both achieved MCICs (-16.2 mm and -11.8 pts respectively).

Table 13. Change in VAS and ODI (Study 3)

	T1	T2	T3
VAS (mm)	33.4±23.3	36.3±22.8	20.1±14.7
ODI (pts)	26.7±11.2	27.8±9.4	16.0±13.5*

Note: * Indicates significant pairwise comparison between T1 and T3

4.3.2 Discussion

The purpose this study was to examine the effects of a 12 week ILEX resistance training intervention in participants with chronic LBP upon indirect determination of disc hydration through spinal height measured using seated stadiometry. To the author's knowledge this is the first study to examine whether positive changes in the discs measured *in vivo* result from exercise interventions in participants with chronic LBP.

Symptomatic degenerative discs show a number of abnormalities including reduced glycosaminoglycans, dehydration, and reduced nucleus pulposus pH (Kitano et al. 1993). Some have suggested that metabolic abnormalities in the intervertebral disc might be improved, thus potentially halting or reversing the degenerative process, through appropriate exercise of the lumbar spine (Mooney et al. 2006; Norris, 2008; Mayer et al. 2008). The exercise specifically considered by Mooney et al. (2006) and Mayer et al. (2008) was ILEX. Not all exercises are equally effective in conditioning the lumbar extensors and ILEX has been suggested here as optimal for this purpose (Steele et al., 2013^o). Thus it may offer potential for improving disc condition also.

Some studies have suggested that continued compressive loading can contribute to harmful responses in the disc in a dose-dependent manner (i.e. magnitude and duration), which might further suggest cause for concern in employing ILEX resistance exercise for those with LBP (Lotz et al., 1998; Kroeber et al., 2002). However, this dose-dependent

mechanism has important implications for ILEX resistance exercise, which is also typically employed in a dose-dependent manner. As noted, ILEX rehabilitation selects a resistance that allows only ~8-12 repetitions and exercise is performed to momentary muscular failure using this resistance, which has been suggested as optimal for strength and hypertrophic adaptations (Fisher et al., 2011; 2013). An exercise frequency of once per week has also been identified as sufficient for improving lumbar extension strength, pain and disability (Graves et al., 1990^b; Bruce-Low et al., 2012). Thus ILEX rehabilitation represents a relatively high loading on the disc though at a low frequency and volume. Walsh and Lotz (2004) report that, in comparison to higher frequency and lower load compression, lower frequency and higher load compression induces positive improvements in disc proteoglycan content, matrix gene expression and rate of cell apoptosis. Thus there may be potential for ILEX rehabilitation to exert a similar adaptive effect. Indeed, Maclean et al. (2004; 2005) have also showed that anabolic and catabolic responses in the nucleus are dependent upon load and frequency with anabolic genes being stimulated at low frequencies and catabolic genes being stimulated at higher frequencies. They also revealed that very low loading had no effect upon gene expression suggesting that some degree of loading, though at a low frequency, is required to stimulate an adaptive anabolic response.

These studies have examined what might be considered regenerative processes, but as we have highlighted, a loss of disc hydration is also present in degenerative discs (Kitano et al., 1993) and so rehydration may also be an important consideration. Ferguson et al. (2004) have shown that loading increases fluid flow across the disc, which in turn also enhances transport of larger solutes into the intervertebral disc. ILEX rehabilitation may enhance pressure variance across the disc through its flexion-extension cycles and thus enhance interstitial fluid flow (Bruce-Low et al., 2012). The findings of Ferguson et al. (2004) would lend biological plausibility to this potential mechanism also. Further, Wang et

al. (2007) have presented that while static loading contributes to catabolic activity, dynamic compressive loading contrastingly promotes anabolic activity.

Research thus far has been conducted using *in vitro* animal models. This study is apparently the first to attempt to examine the chronic effects of specific loading upon the disc *in vivo*. Due to suggestions from other authors regarding use of ILEX to 'rehydrate' the discs (Norris, 2008; Mayer et al., 2008) and that loading increases fluid flow, enhancing transport of larger solutes into the intervertebral disc (Ferguson et al., 2004), it was considered that ILEX may create pressure variance across the disc through flexion-extension cycles and thus enhance interstitial fluid flow. Thus it was hypothesised a 12 week ILEX resistance training intervention in chronic LBP participants would improve disc hydration as measured indirectly through spinal height measures using seated stadiometry.

This study however failed to reject the null hypothesis and suggested that, although the 12 week intervention improved ILEX strength, pain and disability, there was no change in any of the seated stadiometry variables measured. Seated stature measures did not achieve significance and were also within the between-day range of error determined for the custom seated stadiometry set-up used (See appendix 7.6). As no other study has examined the effects of an intervention upon chronic adaptation in the discs *in vivo* it is not possible to discern whether these results truly reflect a lack of change from the intervention or whether they stem from the testing utilised.

Acute studies of stature changes from various loading conditions reveal a wide range of changes some of which the current set-up used may have been sensitive enough to detect; ~0.5mm (Healey et al., 2004), to ~3mm (Owens et al., 2009), ~5mm (Kourtis et al., 2004), ~7.5 - 10mm (Magnusson et al., 1996), and ~6-7mm (Rodacki et al., 2007). Considering the possible magnitudes of acute differences detected by some of these

studies, it may be that the ILEX intervention merely did not induce any change in hydration of the discs, or at least not of a sufficient magnitude to be detected. MRI is more sensitive in detecting changes in disc hydration, in particular due to the ability to examine individual discs, as opposed to the cumulative total of their height, including the vertebral bodies and other osseoligamentous structures, when using seated stadiometry. Kourtis et al. (2004) report an error when using MRI of ~0.5mm which is considerably lower than the error within our custom seated stadiometry set-up (3.1mm). Further study should examine whether changes in disc hydration occur from exercise based interventions when tested using MRI. Also, loss of hydration is only one aspect of a range of possible factors indicating disc condition (Adams & Roughley, 2006) and so, though there may not be a change in disc hydration after exercise interventions, the potential mechanisms of adaptation might impart positive adaptation in other features of the disc. Additional categorisation of disc condition would be a further benefit of follow-up study utilising MRI.

A further aspect examined in the present study was the time dependent loss of stature, or shrinkage, related to spinal loading. This is considered an indicator of spinal 'creep' due to its viscoelastic properties and may reflect the potential for spinal structures to experience time related changes in biomechanical stresses (Magnusson et al., 1990; Van Dieen & Toussaint, 1993). Indeed stature shrinkage from constant static loading differs between asymptomatic controls and chronic LBP participants (Kanlayanaphotporn et al., 2003). This study examined change in spinal height over a 3 – 3.5 minute test where the participant remained seated in the stadiometer. The between-day reliability of this variable in our custom set-up was similar to that reported by others (Kanlayanaphotporn et al., 2002). However, as with measurements of stature, there was no change in shrinkage after the ILEX intervention.

Despite absence of changes in seated stadiometry variables in response to the intervention, changes were observed for ILEX strength, pain and disability. No changes in

any variables were found over the 12 week control period. However, ILEX strength increased significantly over the intervention period and to a similar degree (~34%) as other studies utilising the same intervention (Smith et al., 2011; Bruce-Low et al., 2012). These results also indicated the ILEX intervention period resulted in a significant reduction in disability measured using the ODI between baseline (T1) and re-test after the intervention period (T3). Though change in pain and disability over the intervention period did not achieve significance they were similar to other studies utilising the same ILEX intervention in chronic LBP participants (Smith et al., 2011; Bruce-Low et al., 2012) and thus likely reflect the studies small sample size and thus a type II error. Despite this however, change in pain and disability across the intervention period using VAS and ODI did both achieve MCICs (reduction of ~16 mm and ~12 pts respectively) and therefore can be considered meaningful.

As noted, the testing utilised in this study is a limitation as it may not have been sufficiently sensitive to detect changes in response to the intervention. However, it may be that sample size affected comparisons also. The sample may not have been large enough to detect change in seated stadiometry variables. Thus future study, in addition to considering utilisation of MRI to detect *in vivo* changes in disc condition, should also utilise a larger sample size to account for potential type II error in the present study.

In conclusion, the results of the present study, though further supporting the use of ILEX resistance training to improve ILEX strength, pain and disability, did not find any effect upon spinal height measures using seated stadiometry. Thus, despite its impact upon other aspects of the multifactorial nature of LBP, suggestion that ILEX exercise improves disc condition in chronic LBP participants is presently not supported and remains a speculative hypothesis requiring further study.

5. CONCLUSIONS

Regarding research on LBP and its treatment modalities, Helmhout and colleagues (2004^b) have highlighted that,

*“...the majority of studies on LBP management consist of multimodal interventions, which include physical, behavioural, educational and/or ergonomic elements...to obtain a better view on the (relative) efficacy of either of these concepts, **unimodal intervention programs** like ours²⁶ need to be evaluated.”*

They go on further to note that *“...exercise as the primary entrance for restoring back function **has a wide span of treatment effects**, including improvements for cognitive and/or behavioural variables.”*

Within this thesis these points have been considered and it would seem that this is the first to specifically examine the area in this manner. The use of a single intervention, consisting of a minimal ILEX resistance training intervention, has allowed direct inference as to whether this specific exercise approach does indeed confer a wide span of treatment effects considering the multifactorial nature of dysfunction in LBP.

To reiterate, the following studies were proposed in the introduction and the accompanying hypotheses to be tested.

- **Study 1** - Can ILEX resistance training through a limited ROM can produce strength gains throughout the full ROM in symptomatic chronic LBP participants?
 - **Hypothesis 1** - Limited ROM ILEX resistance training will produce full ROM strength improvement in chronic LBP participants in addition to

²⁶ Helmhout et al. (2004^b) are referring to ILEX here.

improving ROM and reducing pain and disability with no difference between limited or full ROM exercise.

- **Study 2** - Can ILEX resistance training effect gait variability in chronic LBP participants?
 - **Hypothesis 2** - ILEX resistance training will produce reductions in gait variability in chronic LBP participants.
- **Study 3** - Can ILEX resistance training effect inter-vertebral disc hydration, measured via seated stadiometry, in chronic LBP participants?
 - **Hypothesis 3** - ILEX resistance training will produce improvements in intervertebral disc hydration measured indirectly via seated stadiometry.

Of these, the first two hypotheses were found to be supported through the studies conducted whereas the final hypothesis was refuted. Discussion of the specific results was presented in the previous chapter; however, it appears that this thesis has also provided general support for its initial premise.

- *Initial Premise* - Lumbar extensor deconditioning is implicated as a cause of low back injury and LBP, may influence other associated physical symptoms, dysfunctions and pain causing mechanisms in LBP, and in the majority of cases of LBP and chronic LBP may be a predominant causative factor (though it is not implied to be causative in all cases). The corollary of this being that exercise aimed at addressing this (i.e. ILEX resistance training) is a justified approach to preventing and treating LBP and chronic LBP and may affect other symptoms and dysfunctions yet to be examined.

Though the final study did not support this, both the first and second studies demonstrated that the use of ILEX to address the lumbar extensor deconditioning found in LBP offers multifactorial effects. Indeed its effectiveness is maintained even in consideration of the

associated dysfunction of limited ROM, and further benefit was demonstrated in improving the commonly associated gait dysfunction found in LBP.

The value of a single minimal intervention capable of providing a range of benefits in treating LBP, such as ILEX, is considerable. The use of ILEX for treatment of LBP should be seriously considered by those providing such service. This should involve weighing of its costs against its benefits, including: efficacy in improving lumbar extensor condition, reduction in pain and disability, reduction of treatment time through its minimal approach, and any additional benefits (including those presented here and any that may be reported in the future) when making decisions in this regard (Helmhout et al., 2008^a). Though many exercise-based interventions for LBP are often found to provide statistically significant improvements, for many the magnitude of their effect size is found to be quite small (Hayden et al., 2005). Considering effectiveness of interventions based purely on frequentist approaches using non-magnitude based statistical inference (i.e. Neyman-Pearson significance testing) has received considerable criticism over recent years (Wilkinson, 2013). Consideration of the use of ILEX from a Bayesian-style approach, taking into account prior estimates of low effect size magnitudes from other exercise interventions (Hayden et al., 2005), and considering the large effect sizes reported for ILEX here, provides further suggestion of the value it might present. Improvements in pain and disability for each of the studies conducted here met MCICs (Ostelo et al., 2008) and effect sizes were large (0.9 to 1.67 for VAS and 1.42 to 2.28 for ODI; Cohen, 1992) and comparable to other recent studies using ILEX (Smith et al., 2011; Bruce-Low et al., 2012).

As noted in the introduction, the costs associated with LBP are enormous and stem from both direct costs involving treatment and indirect costs from loss of productivity. It has already been demonstrated that ILEX can help to reduce these costs. ILEX is cost effective when compared to other treatment approaches, including surgery, especially

through reducing re-utilisation of the health-care system. However, that there is potentially a range of dysfunctions that can be addressed through use of ILEX in LBP there is even greater potential for cost reduction. Single interventions are likely to be considerably more cost effective in comparison to complex multidisciplinary interventions involving multiple service providers (Breen et al., 2006). Redirection of treatment towards an active single treatment approach considering LBP as a condition with a number of multifactorial dysfunctions, which might be improved through such treatment, should be considered.

A minimal approach such as ILEX also offers the benefit of time efficiency. ILEX sessions require at least ~50% less time compared to regular physical therapy (Helmhout et al., 2008^b). Recent analysis suggested greater benefit may occur with a greater frequency of exercise sessions (an additional eight sessions required to improve VAS scores by 1mm compared to controls [Ferreira et al., 2010]). ILEX specifically, however, is highly effective using only a single weekly session with no further benefit from additional sessions (Bruce-Low et al., 2012). It seems that ILEX is also as effective as a single intervention approach (Risch et al., 1993; Holmes et al., 1996; Al-Obaidi et al., 2005; Carlson & MacKay, 2010; Smith et al., 2011; Bruce-Low et al., 2012) and that the benefits can occur from as little as one session per week taking approximately 10-15 minutes with only 1-2 minutes of that comprising exercise. As one of the biggest economic losses through chronic LBP is due to work hours lost, both through treatment and absenteeism, a workplace strengthening program (Mooney et al., 1993; Mooney et al., 1995; Matheson & Mooney, 2007; Dreisinger, 2000) using ILEX could be a promising occupational approach.

Considering this the following recommendation could be made for practical implementation of ILEX as a widely offered treatment approach for LBP. As presently ILEX is not a widely available approach, more service providers should consider investing in equipment that might allow it to be performed. A common argument against this recommendation is that initial cost of purchasing such equipment (Smith et al., 2008) and

depreciation costs of materials (Slade & Keating, 2006) are high. Yet there are now a number of ILEX devices available ranging in price (e.g. Lumbar Extension Machine, MedX, Ocala, Florida; BackUp Dynamometer, Priority One Equipment, Grand Junction, Colorado; Lower Back Revival System, OriGENE Concepts BV, Delft, the Netherlands etc.). Some offer sophisticated testing options (such as the MedX Lumbar Extension Machine) whereas others are purely for exercise use. Although sophisticated testing might be desirable in research it may be less of a concern to clinicians and so more 'low tech' options might be considered. Despite this, even the cost of more expensive ILEX devices is low in comparison to the costs associated with LBP (Smith et al., 2008). As such service providers such as clinicians, public and private occupational health departments, and public gym facilities should consider acquisition of ILEX devices in order to make it more widely available to those suffering from LBP.

5.1 Future Directions

Despite the novel findings presented here, as with any process of scientific enquiry these hypotheses require further attempts at refutation through experimentation, considering potential limitations of the methodologies used here, which may or may not be found to support them. In addition, in discussion of the findings reported here further questions also pose themselves from which hypotheses might be generated and tested in future work. The following summarises some of the key questions that arose in discussing the findings of this work and brief suggestions of how they might be examined.

5.1.1 Study 1

- Though ROM did not improve for either full or limited ROM training this may have been due to participants in this study not having sufficiently impaired ROM compared with other studies of ILEX (Nelson et al., 1995; Bruce-Low et al., 2012). Thus, although limited ROM training does not differ from full ROM training with respect to full ROM ILEX strength, pain, or disability, future research might

examine whether limited ROM training differs compared to full ROM training with respect to changes in ROM. This research should therefore preferably identify participants with more severely impaired ROM.

- Recent systematic review has suggested (Steiger et al., 2012^a) that changes in muscular performance factors do not correlate with changes in pain and disability thus bringing doubt to claims regarding these factors as mechanisms for improvement. However, these claims and the specificity of the exercises examined in this review have recently been questioned (Steele & Bruce-Low, 2012). Steiger et al., (2012^a) did not include studies examining ILEX resistance training in their review, nor did they include studies examining ILEX strength as a muscular performance outcome. Thus their results might instead reflect the lack of specificity of the exercise interventions they examined and the fact that they did not specifically look at lumbar extensor condition as an outcome. This study therefore, as it included sufficient sample size, examined correlations between change in ILEX strength, pain and disability finding that greater increases in strength were associated with greater improvements in pain and disability. Nelson et al., (1995) also reported similar correlations specifically looking at ILEX strength in a large sample. However, as Steiger et al., (2012^a) noted, numerous studies have not reported correlations thus rendering difficulty in properly assessing this association. Future research should then ensure reporting of these correlations and a valuable area of investigation might be to perform further systematic review, attempting to obtain raw data from previous studies of ILEX resistance training in LBP, in order to calculate correlations from a larger data set to compare with those reported by Steiger et al. (2012^a). This, along with further comparative intervention studies, would add to the checklist of evidence required by the Austin Bradford Hill criteria (Hill, 1965) for determining a causative relationship between muscular factors and LBP. It would also offer further information regarding the 'Black Box' of

treatment mechanisms underlying exercise approaches to chronic LBP (Helmhout et al., 2008^a).

- Avoidance of full extension and flexion may have a significant impact upon pain experienced by symptomatic participants (Donelson et al., 1991), and avoiding painful positions through use of directional preference based movement impacts adherence to exercise (Long et al., 2004). Thus limited ROM exercise might affect adherence positively in comparison to full ROM exercise. Our results did not support this; however, differences in adherence may present themselves over a longer term intervention. Alternatively ILEX may instead be an approach that maintains adherence thus making it an even more valuable intervention approach. Future work should implement longer intervention periods (>12 weeks) to examine whether adherence is maintained and if differences in limited and full ROM exercise manifest.
- Similarly, limited ROM ILEX exercise may be safer for chronic LBP participants as it avoids positions deemed close to the margin of safety for mechanical injury to occur (Van Dieen et al., 1998; Dolan & Adams, 1998; Sparto et al., 1997^a; Sparto et al., 1997^b). In the longer study proposed above differences in acute injury during exercise could be recorded to examine whether a difference based upon this hypothesis indeed does occur.

5.1.2 Study 2

- Lumbar kinematic analysis during gait in this study involved the use of novel methods of calculating the variability in the waveform independent of factors that might affect the mean offset variability in the data (O'Dwyer et al., 2009). Prior work which examined participants with LBP utilised Winter's CV, which does not consider this, however, has shown greater variability compared to healthy asymptomatic participants (Vogt et al., 1999; Vogt et al., 2001). Though the present data using Winter's CV suggest that the sample of chronic LBP

participants did indeed show greater variability compared to asymptomatic participants in earlier research, CV_p has not been examined in this population. Thus to clarify whether variability in waveform patterns in participants with chronic LBP is indeed greater than healthy asymptomatic participants further research should examine this in the latter population.

- Baseline data from this study demonstrated significant correlation between reduced ILEX strength and both greater transverse plane variability and smaller transverse displacements. However, transverse plane coordination of the trunk and pelvis, and variability in the phase differences has also been examined (Lamoth et al., 2002; Lamoth et al., 2006^a; van der Hoorn et al., 2012). Transverse movement is more rigid in chronic LBP, yet within the smaller range of movement there is poor waveform pattern repeatability. The rigidity seen in trunk/pelvis transverse kinematic coordination in chronic LBP (Lamoth et al., 2002; Lamoth et al., 2006^a; van der Hoorn et al., 2012) may still be a compensatory manifestation of lumbar extensor deconditioning. Considering this it may be of future interest to examine the relationship between ILEX and trunk/pelvis coordination in those with chronic LBP.
- Though baseline data indicated a relationship between transverse CV_p and ILEX strength, the intervention significantly reduced sagittal plane CV_p . This was proposed as potentially due to the plane of movement that ILEX is performed in (sagittal). Thus future research should examine whether plane of movement specific training may produce consequent plane of movement specific changes in control of the lumbar spine during gait. For example, whether torso rotation may perhaps improve transverse CV_p .

5.1.3 Study 3

- This study found no changes in either spinal height or spinal shrinkage measured using seated stadiometry and thus suggested the ILEX intervention did not

produce any improvements in disc hydration or viscoelastic properties of the spine. However, as noted it may have been that the testing utilised was not of sufficient sensitivity or reliability to detect a change resulting from the intervention. In addition, a range of possible factors indicating disc condition exist (Adams & Roughley, 2006). Thus, though disc hydration may not change after exercise interventions, ILEX might instead impart positive adaptation in other disc features. Further study utilising MRI to detect changes in disc hydration, in addition to other aspects of disc condition in vivo, also utilising a larger sample size and preferably randomised controlled trial design, should be conducted.

5.1.4 General

- Though in this thesis four aspects of physical dysfunction in LBP have been focused upon in particular (lumbar extensor deconditioning, limited ROM, gait variability, disc degeneration), future work should examine the role that lumbar extensor deconditioning plays within LBP with respect to other dysfunctions. Longitudinal prospective study might better identify the role that lumbar extensor deconditioning, in the form of reduced ILEX strength/endurance, atrophy, or excessive fatigability, plays in low back injury and development of LBP along with the other physical dysfunctions associated with it including both development of physical and psycho-social dysfunctions. Research along these lines might better clarify whether or not lumbar extensor deconditioning is indeed responsible for the initiation of LBP and the development and progression of its related dysfunctions.
- In addition, further research in those with chronic LBP utilising ILEX as an intervention, yet examining other aspects of dysfunction associated with LBP, would further clarify the role that addressing lumbar extensor deconditioning plays in management of LBP and its related dysfunctions. A multifactorial framework should be utilised to conceptualise this and produce further hypotheses.

- One hypothesis resulting from this conceptualisation is that, once LBP is initiated and dysfunctions are present, whether their presence affects subsequent success from use of interventions such as ILEX. Pre sub-categorisation of participants based upon dysfunctions and then examination of outcomes after ILEX in sub-groups based upon this would allow identification of whether a particular array of dysfunctions might respond best. Nelson et al., (1995) have already demonstrated that ILEX may be effective across a range of broad diagnoses and so ILEX may be found to be broadly effective across a range of dysfunctions.
- Finally, future research needs to better examine comparative approaches to ILEX. There is lack of research comparing ILEX with other exercise modalities which are designed to address other aspects of dysfunction in LBP. For example, stability based training programs are often used to retrain the motor control aspects, while general exercise programs are suggested to mainly affect emotional or psychosocial factors. Carefully conducted research comparing different approaches, with different proposed mechanisms of action, might provide greater understanding into the 'Black Box' of treatment mechanisms, and thus the role that each of these factors play in LBP, allowing better comparison of the efficacy of different approaches (Hemlhout et al., 2008^a). Adding to this is the concern of placebo effects from the use of ILEX as none have yet looked to examine this. Indeed, this is a possible explanation for the positive outcomes reported in this thesis. Risch et al., (1993) have shown that there are psychological effects from ILEX in those with LBP thus it would not be surprising that there might be non-physical mechanisms by which pain and disability are improved through ILEX. Conducting placebo controlled trials in exercise interventions is difficult (Dvir, 2007; Helmhout et al., 2004^b; 2008^b). However, there is the possibility of testing the role that muscular conditioning has upon LBP independent of any placebo type effects through implementation of a 'sham therapy' controlled trial. Participants and researchers conducting the testing could be blinded to the presence of different

groups and participants informed that they will undergo a training program for their lower back without other details (avoiding issue's highlighted in the study by Helmhout et al., [2004^a] regarding participant expectations). One arm of the trial could implement the usual ILEX intervention (1x/week, one set using 80% max TFT, performed to MMF using a controlled repetition duration) and another could implement a 'sham therapy' designed to have little impact upon the musculature (using a low load and avoiding MMF; Steele et al., 2013^b).

6.0 REFERENCES

1. Adams, M. A., A. F. Mannion, and P. Dolan, 1999. Personal risk factors for first-time low back pain. *Spine*. 24(23), pp 2497 – 2505
2. Adams, M. A., and W. D. Hutton, 1985. Gradual disc prolapse. *Spine*. 10, pp 524
3. Adams, M. A., M. Stefanakis, and P. Dolan, 2010. Healing of a painful intervertebral disc should not be confused with reversing disc degeneration: implications for physical therapies for discogenic back pain. *Clinical Biomechanics*. 25, pp 961 – 971
4. Adams, M. A., Roughley, P. J., 2006. What is intervertebral disc degeneration, and what causes it? *Spine*. 31(18), 2151–2161
5. Adams, P., and H. Muir, 1976. Qualitative changes with age of proteoglycans of human lumbar discs. *Annals of Rheumatic Diseases*. 35, pp 289
6. Addison, R., and A. Shultz, 1980. Trunk strength in patients seeking hospitalization for chronic low back disorders. *Spine*. 5(6), pp 539 - 544
7. Alaranta, H., H. Hurri, M. Heliovaara, A. Soukka, and R. Harju, 1994. Non-dynamometric trunk performance tests: reliability and normative data. *Scandinavian Journal of Rehabilitation Medicine*. 26(4), pp 211 215
8. Alaranta, H., K. Tallroth, A. Soukka, and M. Heliovaara, 1993. Fat content of lumbar extensors muscles and low back disability: a radiographic and clinical comparison. *Journal of Spinal Disorders*. 6(2), pp 137 – 140
9. Albert, H. B., and C. Manniche, 2007. Modic changes following lumbar disc herniation. *European Spine Journal*. 16, pp 977-982
10. Al-Obaidi, S. M., P. Beattie, B. Al-Zoabi, and S. Al-Wekeel, 2005. The relationship of anticipated pain and fear avoidance beliefs to outcome in patients with chronic low back pain who are not receiving workers compensation. *Spine*. 30(9), pp 1051 – 1057

11. American College of Sports Medicine, 2009. Progression models in resistance training for healthy adults. *Medicine and Science in Sports and Exercise*. 41(3), pp 687-708
12. Anders, C., H. C. Scholle, H. Wagner, C. Puta, R. Grassme, and A. Petrovitch, 2005. Trunk muscle co-ordination during gait: relationship between muscle function and acute low back pain. *Pathophysiology*. 12 (4), pp 243 – 247
13. Andersson, G. J., 1999. Epidemiological features of chronic low back pain. *The Lancet*. 354(9178), pp 581 – 585
14. Apkarian, A. V., M. C. Bushnell, R. Treede, and J. Zubieta, 2005. Human brain mechanisms of pain perception and regulation in health and disease. *European Journal of Pain*. 9, pp 463 – 484
15. Arendt-Nielsen, L., T. Graven-Nielsen, H. Svarrer, and P. Svensson, 1996. The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. *Pain*. 64(2), pp 231 – 240
16. Arjunan, S. P., D. K. Kumar, W. M. Poon, H. Rudolph, and Y. Hu, 2010. Variability in surface electromyogram during gait analysis of low back pain patients. *Journal of Medical and Biological Engineering*. 30(3), pp 133 - 138
17. Arokoski, J. P. A., M. Kankaanpaa, T. Valta, I. Juvonen, J. Partanen, S. Taimela, K. A. Lindgren, and O. Airaksinen, 1999. Back and hip extensor muscle function during therapeutics exercises. *Archive of Physical Medicine and Rehabilitation*. 80, pp 842 – 850
18. Arokoski, J. P., T. Valta, M. Kankaanpaa, and O. Airaksinen, 2004. Activation of lumbar paraspinal and abdominal muscles during therapeutic exercises in chronic low back pain patients. *Archives of Physical Medicine and Rehabilitation*. 85, pp 823 – 832

19. Arokoski, J. P., T. Valta, O. Airaksinen, and M. Kankaanpää, 2001. Back and abdominal muscle function during stabilization exercises. *Archives of Physical Medicine and Rehabilitation*. 82(8), pp 1089 – 1098
20. Bagnall, K. M., D. M. Ford, K. D. McFadden, B. J. Greenhill, and V. J. Raso, 1984. The histochemical composition of human vertebral muscle. *Spine*. 9(5), pp 470–473
21. Bahr, R., S. O. Andersen, S. Loken, B. Fossan, T. Hansen, and I. Holme, 2004. Low back pain among endurance athletes with and without specific back loading – a cross sectional survey of cross country skiers, rowers, orienteers, and nonathletic controls. *Spine*. 29(4), pp 449–454
22. Bajek, S., D. Bobinac, G. Bajek, T. S. Vranic, B. Lah, and D. M. Dragojevic, 2000. Muscle fiber type distribution in multifidus muscle in cases of lumbar disc herniation. *Acta Medica Okayama*. 54(6), pp 235–241
23. Balague, F., A. F. Mannion, F. Pellise, C. Cedraschi, 2012. Non-specific low back pain – Author’s reply. *Lancet*. 379, pp1874 – 1875
24. Balague, F., P. Damidot, M. Nordin, M. Parnianpour, and M. Waldburger, 1993. Cross-sectional study of the isokinetic muscle strength among school children. *Spine*. 18(9), pp 1199 – 1205
25. Barento, A., M. Hellström, C. G. Cederlund, R. Nyman, and L. Sward, 2009. Back pain and MRI changes in the thoraco-lumbar spine of top athletes in four different sports: a 15 year follow-up study. *Knee Surgery, Sports Traumatology, Arthroscopy*. 17, pp 1125 – 1134
26. Barker, K. L., D. R. Shamley, D. Jackson, 2004. Changes in cross-sectional area of multifidus and psoas in patients with unilateral back pain: the relationship to pain and disability. *Spine*. 29(22), E515–519
27. Barrero, L. H., Y. Hsu, H. Terwedow, M. Perry, J. Dennerlein, J. Brain, and X. Xu, 2006. Prevalence and Physical Determinants of Low Back Pain in a Rural Chinese Population. *Spine*. 31(23), pp 2728 – 2734

28. Bates, B. T., J. S. Dufek, and H. P. Davis, 1992. The effect of trial size on statistical power. *Medicine and Science in Sports and Exercise*. 24(9), pp 1059 – 1068
29. Bates, T. B., C. R. James, and J. D. Dufek, 2004. Single subject analysis. In. N. Stergiou. *Innovative Analyses of Human Movement: Analytical Tools for Human Movement Research*. Champaign: Human Kinetics, pp 3 – 28
30. Batti'e, M., S. J. Bigos, L. D. Fisher, T. H. Hansson, M. E. Jones, and M. D. Wortley, 1989. Isometric lifting strength as a predictor of industrial back pain reports. *Spine*. 14(8), pp 851–856
31. Bayramoglu, M., M. N. Akman, S. Kilinc, N. Cetin, N. Yavuz, and R. Ozker, 2001. Isokinetic measurement of trunk muscle strength in women with chronic low back pain. *American Journal of Physical & Medical Rehabilitation*. 80(9), pp 650 – 655
32. Beattie, P., J. M. Rothstein, and R. L. Lamb, 1987. Reliability of the attraction method for measuring lumbar spine backward bending. *Physical Therapy*. 67(3), pp 364 – 369
33. Behm, D. G., A. M. Leonard, W. B. Young, W. A. Bonsey, and S. N. MacKinnon, 2005. Trunk muscle electromyographic activity with unstable and unilateral exercises. *Journal of Strength & Conditioning Research*. 19(1), pp 193 – 201
34. Beimborn, D. S., and M. C. Morrissey, 1988. A review of the literature related to trunk muscle performance. *Spine*. 13(6), pp 655 – 660
35. Bembien, M. G., K. J. Grump, and B. H. Massey, 1988. Assessment of technical accuracy of the Cybex II dynamometer and analog recording system. *Journal of Orthopaedic and Sports Physical Therapy*. 10, pp 12 – 17
36. Benson, M. E., D. R. Smith, and R. F. Bybee, 2002. The muscle activation of the erector spinae during hyperextension with and without the pelvis restrained. *Physical Therapy in Sport*. 3(4), pp 165 – 174

37. Biedermann, H. J., G. L. Shanks, W. J. Forrest, and J. Inglis, 1991. Power spectrum analyses of electromyographic activity. Discriminators in the differential assessment of patients with chronic low back pain. *Spine*. 16(10), pp 1179 – 1184
38. Biering-Sorensen, F., 1984. Physical measurements as risk indicators for low-back trouble over a one-year period. *Spine*. 9(2), pp 106 – 119
39. Bigos, S. J., D. M. Spengler, N. A. Martin, J. Zeh, L. Fisher, A. Nachemson, and M. H. Wang, 1986. Back injuries in industry: A retrospective study II. Injury factors. *Spine*. 11(3), pp 246–251
40. Boden, S. D., D. O. Davis, T. S. Dina, N. J. Patronas, and S. W. Wiesel, 1990. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. *Journal of Bone & Joint Surgery*. 72, pp 403 – 408
41. Bogduk, N., 1997. Low Back Pain. In. N. Bogduk. *Clinical Anatomy of the Lumbar Spine and Sacrum*. 3rd edition. New York: Churchill Livingstone, pp 187 – 214
42. Bogduk, N., and L. T. Twomey, 1987. *Clinical Anatomy of the Lumbar Spine*. Edinburgh: Churchill Livingstone
43. Bonmati, A., A. Gomez-Olivencia, J. L. Arsuaga, J. M. Carretero, A. Gracia, I. Martinez, C. Lorenzo, J. M. Bermudez de Castro, and E. Carbonell, 2010. Middle Pleistocene lower back and pelvis from an aged human individual from the Sima de los Huesos site, Spain. *Proceedings of the National Academy of Sciences of the United States of America*. 107(43), pp 18386 – 18391
44. Bono, C. M., 2004. Low back pain in athletes. *Journal of Bone and Joint Surgery*. 86(2), pp 382–396
45. Borenstein, D. G., 2000. Epidemiology, etiology, diagnostic evaluation, and treatment of low back pain. *Current Opinion in Orthopedics*. 11(3), pp 225 – 231
46. Borenstein, D. G., J. O'Mara, S. D. Boden, W. C. Lauerman, A. Jacobson, C. Platenberg, D. Schellinger, and S. W. Wiesel, 2001. The value of magnetic

- resonance imaging of the lumbar spine to predict low back pain in asymptomatic subjects. *Journal of Bone & Joint Surgery*. 83(9), pp 1306 – 1311
47. Bortz II, W. M., 1984. The disuse syndrome. *The Western Journal of Medicine*. 141(5), pp 691 – 694
 48. Bouche, K. G. W., O. Vanovermeire, V. K. Stevens, P. L. Coorevits, J. J. Caemaert, D. C. Cambier, K. Verstraete, G. G. Vanderstraeten, and L. A. Danneels, 2011. Computed tomographic analysis of the quality of trunk muscles in asymptomatic and symptomatic lumbar discectomy patients. *BMC Musculoskeletal Disorders*. 12, pp 65
 49. Bouissou, P., P. V. Estrade, F. Goubel, C. Y. Guezennec, and B. Serrurier, 1989. Surface EMG power spectrum and intramuscular pH in human vastus lateralis muscle during dynamic exercise. *Journal of Applied Physiology*. 67(3), pp 1245–1249
 50. Bourke, J. B., 1971. The paleopathology of the vertebral column in ancient Egypt and Nubia. *Medical History*. 15(4), pp 363 – 375
 51. Bousema, E. J., J. A. Verbunt, H. A. Seelen, J. W. Vlaeyen, and J. A. Knottnerus, 2007. Disuse and physical deconditioning in the first year after the onset of back pain. *Pain*. 130(3), pp 279–286
 52. Boyce, R., E. Boone, J. Stallings, and C. Wilde, 2008. A multidisciplinary approach to a time-efficient low back exercise intervention in a small manufacturing plant: A case study. *Journal of Exercise Physiology Online*. 11(4), pp 12 – 24
 53. Breen, A. C., M. W. Van Tulder, B. W. Koes, I. Jensen, R. Reardon, and G. Bronfort, 2005. Mono-disciplinary or multidisciplinary back pain guidelines? How can we achieve a common message in primary care? *European Spine Journal*. 15(5), pp 641 – 647
 54. Brinkmann, P., 1985. Pathology of the vertebral column. *Ergonomics*. 28(1), pp 77 – 80
 55. Bruce-Low, S., D. Smith, S. Burnet, J. Fisher, G. Bissell, and L. Webster, 2012. One lumbar extension training session per week is sufficient for gains and reductions in

- pain in patients with chronic low back pain ergonomics. *Ergonomics*. 55(4), pp 500 – 507
56. Butler, D., J. H. Trafimow, G. B. Andersson, T. W. McNeil, and M. S. Huckman, 1990. Discs degenerate before facets. *Spine*. 15(2), pp 111 – 113
57. Cady, L. D., D. P. Bischoff, E. R. O'Connell, P. C. Thomas, and J. H. Allan, 1979. Strength and fitness and subsequent back injuries in firefighters. *Journal of Occupational Medicine*. 21(4), pp 269–272
58. Callaghan, J. P., A. E. Patla, and S. M. McGill, 1999. Low back three-dimensional joint forces, kinematics, and kinetics during walking. *Clinical Biomechanics*. 14, pp 203 – 216
59. Carlson, J., and G. MacKay, 2010. *Impact of specific muscular strength therapy on patients with chronic lower back pain*. Functional Physio. Available: <http://www.functionalphysio.co.nz/therapy>
61. Carpenter, D. M., and B. W. Nelson, 1999. Low back strengthening for the prevention and treatment of low back pain. *Medicine & Science in Sport & Exercise*. 31(1), pp 18 – 24
62. Carpenter, D. M., J. E. Graves, M. L. Pollock, S. H. Leggett, D. Foster, B. Holmes, and M. N. Fulton, 1991. Effect of 12 and 20 weeks of resistance training on lumbar extension torque production. *Physical Therapy*. 71(8), pp 580 – 588
63. Carpes, F. P., F. B. Reinehr, and C. B. Mota, 2008. Effects of a program for trunk strength and stability on pain, low back and pelvis kinematics, and body balance: A pilot study. *Journal of Bodywork and Movement Therapies*. 12, pp 22 – 30
64. Carpinelli, R. N., 2008. The size principle and a critical analysis of the unsubstantiated heavier-is-better recommendation for resistance training. *Journal of Exercise Science & Fitness*. 6(2), pp 67 – 86

65. Carpinelli, R., R. M. Otto, and R. A. Winett, 2004. A critical analysis of the ACSM position stand on resistance training: insufficient evidence to support recommended training protocols. *Journal of Exercise Physiology*. 7, pp 1 – 60
66. Carragee, E. J., C. M. Tanner, S. Khurana, C. Hayward, J. Welsh, E. Date, T. Truong, M. Rossi, and C. Hagle, 2000. The rates of false-positive lumbar discography in select patients without low back symptoms. *Spine*. 25(11), pp 1373 – 1380
67. Carrera-Bastos, P., M. Fontes-Villalba, J. H. O’Keefe, S. Lindeberg, and L. Cordain, 2011. The western diet and lifestyle and diseases of civilization. *Research Reports in Clinical Cardiology*. 2, pp 15 – 35
68. Cassisi, J. E., M. E. Robinson, P. O’Connor, and M. MacMillan, 1993. Trunk strength and lumbar paraspinal muscle activity during isometric exercise in chronic low-back pain patients and controls. *Spine*. 18(2), pp 245 – 251
69. Chaffin, D. B., G. D. Herrin, W. M. Keyserling, 1978. Preemployment strength testing. An updated position. *Journal of Occupational Medicine*. 20(6), pp 403–408
70. Chan, A. C. W., S. J. Ferguson, and B. Gantenbein-Ritter, 2011. The effects of dynamic loading on the intervertebral disc. *European Spine Journal*. 20, pp 1796 – 1812
71. Cheung K. M. C., J. Karppinen, D. Chan, D. W. H. Ho, Y. Song, P. Sham, K. S. E. Cheah, J. C. Y Leong, and K. D. K. Luk, 2009. Prevalence and pattern of lumbar magnetic resonance imaging changes in a population study of one thousand forty-three individuals. *Spine*. 34(9), pp 934–940
72. Choi, G., P. P. Raiturker, M. Kim, C. D. Jin, Y. Chae, and S. Lee, 2005. The effect of early isolated lumbar extension exercise program for patients with herniated disc undergoing lumbar discectomy. *Neurosurgery*. 57, pp 764 – 772

73. Cholewicki, J., M. M. Panjabi, and A. Khachtrayan, 1997. Stabilizing function of trunk flexor-extensor muscles around a neutral spine posture. *Spine*. 22(19), pp 2207–2212
74. Cholewicki, J., and J. J. Van Vliet, 2002. Relative contribution of trunk muscles to the stability of the lumbar spine during isometric exertions. *Clinical Biomechanics*. 17(2), pp 99–105
75. Chulvi-Medrano, I., X. Garcia-Masso, J. C. Colado, C. Pablos, J. A. De Moraes, and M. A. Fuster, 2010. Deadlift muscle force and activation under stable and unstable conditions. *Journal of Strength and Conditioning Research*. 24(10), pp 2723 – 2730
76. Clark, B. C., T. M. Manini, and L. L. Ploutz-Snyder, 2003. Derecruitment of the lumbar musculature with fatiguing trunk extension exercise. *Spine*. 28(3), pp 282 – 287
77. Clark, B. C., T. M. Manini, J. M. Mayer, L. L. Ploutz-Snyder, and J. E. Graves, 2002. Electromyographic activity of the lumbar and hip extensors during dynamic trunk extension exercise. *Archives of Physical Medicine and Rehabilitation*. 83, pp 1547 – 1552
78. Cohen, J., 1992. A power primer. *Psychological Bulletin*. 112(1), pp 155 – 159
79. Colado, J. C., C. Pablos, I. Chulvi-Medrano, X. Garcia-Masso, J. Flandez, and D. G. Behm, 2011. The progression of paraspinal muscle recruitment intensity in localised and global strength training exercises is not based on instability alone. *Archives of Physical Medicine and Rehabilitation*. 92(11), pp 1875 – 1883
80. Contreras, B. M., J. B. Cronin, B. J. Schoenfeld, R. J. Nates, and G. T. Sonmez, 2013. Are all hip extension exercises created equal? *Strength and Conditioning Journal*. 35(2), pp 17-22
81. Cooper, R. G., M. J. Stokes, C. Sweet, R. J. Taylor, and M. I. Jayson, 1993. Increased central drive during fatiguing contractions of the paraspinal muscles in chronic low back pain. *Spine*. 18(5), pp 610-616

82. Cooper, R. G., W. St. Clair Forbes, and M. I. V. Jayson, 1992. Radiographic demonstration of paraspinal muscle wasting in patients with chronic low back pain. *British Journal of Rheumatology*. 31, pp 389 – 394
83. Costa, K., 2010. *Effects of a trunk strengthening program on pain perception, strength and flexibility on patients with non-specific low back pain*. Pindara Physiotherapy and Sports Medicine. Available: <http://pindaraphysio.com.au/?p=127>
84. Crisco, J. J., and M. M. Panjabi, 1991. The intersegmental and multisegmental muscles of the lumbar spine. A biomechanical model comparing lateral stabilizing potential. *Spine*. 16(7), pp 793–799
85. Croft, P. R., G. J. Macfarlane, A. C. Papageorgiou, E. Thomas, and A. J. Silman, 1998. Outcome of low back pain in general practice: a prospective study. *BMJ*. 316(7141), pp 1356 – 1359
86. Crossman, K., M. Mahon, P. J. Watson, J. A. Oldham, and R. G. Cooper, 2004. Chronic low back pain-associated paraspinal muscle dysfunction is not the result of a constitutionally “adverse” fiber-type composition. *Spine*. 29(6), pp 628 – 634
87. Da Fonseca, J. L., M. Magini, and T. H., De Freitas, 2009. Laboratory gait analysis in patients with low back pain before and after a Pilates intervention. *Journal of Sport Rehabilitation*. 18, pp 269 - 282
88. Da Silva, R. A., A. B. Arsenault, D. Gravel, C. Lariviere, and E. De Oliveira, 2005. Back muscle strength and fatigue in healthy and chronic low back pain subjects: A comparative study of 3 assessment protocols. *Archives of Physical Medicine and Rehabilitation*. 86, pp 722 – 729
89. Da Silva, R. A., C. Lariviere, A. B. Arsenault, S. Nadeau, and A. Plamondon, 2009. Pelvic Stabilization and Semisitting Position Increase the Specificity of Back Exercises. *Medicine & Science in Sports & Exercise*. 41(2), pp 435 -443

90. Daeraga, L., A. Edin-Liljegren, and P. Sjolander, 2003. Work-related musculoskeletal pain among reindeer herding Sami in Sweden – a pilot study on causes and prevention. *Circumpolar Health*. 63, Supp 2, pp 343 – 348
91. Dagenais, S., J. Caro, and S. Haldeman, 2008. A systematic review of low back pain cost of illness studies in the United States and internationally. *Spine*. 8(1) pp, 8 – 20
92. Danneels, L. A., G. G. Vanderstraeten, D. C. Cambier, E. E. Witrouw, and H. J. De Cuyper, 2000. CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. *European Spine Journal*. 9, pp 266 – 272
93. Danneels, L. A., G. G. Vanderstraeten, D. C. Cambier, E. E. Witvrouw, J. Bourgois, W. Dankaerts, and H. J. De Cuyper, 2001. Effects of three different training modalities on the cross sectional area of the lumbar multifidus muscle in patients with chronic low back pain. *British Journal of sports Medicine*. 35(3), pp 186 – 191
94. De Luca, C., and R. Merletti, 1988. Surface myoelectric signal cross-talk among muscles of the leg. *Electroencephalography and Clinical Neurophysiology*. 69(6), pp 568–575
95. De Luca, C., 1997. The Use of Surface Electromyography in Biomechanics. *Journal of Applied Biomechanics*. 13, pp 135–163
96. De Luca, C., 1993. Use of the surface EMG signal for performance evaluation of back muscles. *Muscle and Nerve*. 16, pp 210–216
97. De Roos, A., H. Kressel, C. Spritzer, and M. Dalinka, 1987. MR imaging of marrow changes adjacent to end plates in degenerative lumbar disk disease. *American Journal of Roentgenology*. 149(3), pp 531 – 534
98. de Schepper, E. I. T., J. Damen, J. B. J. van Meurs, A. Z. Ginai, M. Popham, A. Hofman, B. W. Koes, and S. M. Bierma-Zeinstra, 2010. The association between lumbar disc degeneration and low back pain. *Spine*. 35(5), pp 531–536
99. De Vet, H. C., C. B. Terwee, R. W. Ostelo, H. Beckerman, D. L. Knol, and L. M. Bouter, 2006. Minimal changes in health status questionnaires: distinction between

minimally detectable change and minimally important change. *Health and Quality of Life Outcomes*. 4, pp 54

100. DeLeo, J. A., and B. A. Winkelstein, 2002. Physiology of chronic spinal pain syndromes: From animal models to biomechanics. *Spine*. 27, pp 2526 – 2537
101. DeLorme, T. L., 1945. Restoration of muscle power by heavy-resistance exercises. *Journal of Bone & Joint Surgery*. 27, pp 645
102. DeLorme, T. L., and A. L. Watkins, 1948. Technics of progressive resistance exercise. *Archives of Physical Medicine and Rehabilitation*. 29(5), pp 263 – 273
103. Demoulin, C., S. Grosdent, I. Debois, G. Mahieu, D. Maquet, B. Jidovsteff, J. L. Croisier, J. M. Crielaard, and M. Vanderthommen, 2006. Inter-session, inter-tester and inter-site reproducibility of isometric muscle strength measurements. *Isokinetics and Exercise Science*. 14(4), pp 317 – 325
104. DesCartes, R., 1664. *L'Homme*. E Angot: Paris
105. Deutsch, F. E., 1996. Isolated lumbar strengthening in the rehabilitation of chronic low back pain. *Journal of Manipulative and Physiological Therapeutics*. 19(2), pp 124 – 133
106. Dolan, P., and M. A. Adams, 1998. Repetitive lifting tasks fatigue the back muscles and increase the bending moment acting on the lumbar spine. *Journal of Biomechanics*. 31(8), pp 713 – 721
107. Donelson, R., G. McIntosh, and H. Hall, 2012. Is it time to rethink the typical course of low back pain? *PM & R*. 4(6), pp 394-401
108. Donelson, R., W. Grant, C. Kamps, and R. Medcalf, 1991. Pain response to sagittal end-range spinal motion. A prospective, randomized, multicentered trial. *Spine*. 16(6), pp S206 – 212
109. Donisch, E. W., and J. V. Basmajian, 1972. Electromyography of deep back muscles in man. *American Journal of Anatomy*. 133(1), pp 25 – 36

110. Downie, A., C. M. Williams, N. Henschike, M. J. Hancock, R. W. Ostelo, H. C. de Vet, P. Macaskill, L. Irwiq, M. W. van Tulder, B. W. Koes, and C. G. Maher, 2013. Red flags to screen for malignancy and fracture in patients with low back pain: a systematic review. *BMJ*. 347, pp f7095
111. Drake, R. L., A. W. Vogl, and A. W. M. Mitchell, 2010. *Gray's Anatomy for Students*. New York: Churchill Livingstone
112. Dreisinger, T. E., 2000. Does prevention work. In: *San Diego Comprehensive Care Symposium*. San Diego CA
113. Drinkwater, E. J., T. W. Lawton, R. P. Lindsell, D. B. Pyne, P. H. Hunt, and M. J. McKenna, 2005. Training leading to repetition failure enhances bench press strength increases in elite junior athletes. *Journal of Strength and Conditioning Research*. 19(2), pp 382 – 388
114. Dul, J., G. E. Johnson, R. Shiavi, and M. A. Townsend, 1984. Muscular synergism II: minimum fatigue criterion for load sharing between synergistic muscles. *Journal of Biomechanics*. 17, pp 675 – 684
115. Dvir, Z., 2007. Muscle performance enhancement in some non-orthopaedic conditions: Evidence based on modified randomised controlled trials. *Isokinetics and Exercise Science*. 15, pp 1-9
116. Edgar, M. A., 2007. The nerve supply of the lumbar intervertebral disc. *Journal of Bone and Joint Surgery*. 89(9), pp 1135 – 1139
117. Ekman, M., O. Johnell, and L. Lidgren, 2005. The economic cost of low back pain in Sweden in 2001. *Acta Orthopaedica*. 76(2), pp 275 – 284
118. Endean, A., K. T. Palmer, and D. Coggon, 2011. Potential of magnetic resonance imaging findings to refine case definition for mechanical low back pain in epidemiological studies. *Spine*. 36(2), pp 160 – 169
119. Engel, G. L., 1980. The biopsychosocial model and the education of health professionals. *Annals of the New York Academy of Sciences*. 310, pp 169-187

120. Escamilla, R. F., A. C. Francisco, A. V. Kayes, K. P. Speer, and C. T. Moorman III, 2002. An electromyographic analysis of sumo and conventional style deadlifts. *Medicine and Science in Sport and Exercise*. 34(4), pp 682 – 688
121. Fairbank, J. C., J. Couper, J. B. Davies, and J. P. O'Brien, 1980. The Oswestry low back pain disability questionnaire. *Physiotherapy*. 66(8), pp 271 – 273
122. Farfan, H. F., 1975. Muscular mechanism of the lumbar spine and the position of power and efficiency. *Orthopaedic Clinics of North America*. 6, pp 135 – 144
123. Farfan, H. F., and S Gracovetsky, 1984. The nature of instability. *Spine*. 9(7), pp 714 – 719
124. Farina, D., R. Merletti, and R. M. Enoka, 2004. The extraction of neural strategies from the surface EMG. *Journal of Applied Physiology*. 96, pp 1486–1495
125. Ferguson, S. J., K. Ito, and L. P. Nolte, 2004. Fluid flow and convective transport of solutes within the intervertebral disc. *Journal of Biomechanics*. 37(2), pp 213 – 221
126. Ferreira, M. L., R. J. E. M. Smeets, S. J. Kamper, P. K. Ferreira, and L. A. C. Machado, 2010. Can we explain heterogeneity among randomized clinical trials of exercise for chronic back pain? A meta-regression analysis of randomized controlled trials. *Physical Therapy*., 90(10), pp 1383 – 1403
127. Fidler, M. W., R. L. Jowett, and J. D. G. Troup, 1975. Myosin ATPase activity in multifidus muscle from cases of lumbar spinal derangement. *Journal of Bone and Joint Surgery*. 57(2), pp 220–227
128. Filler, A. G., 2007. Emergence and optimisation of upright posture among hominiform hominoids and the evolutionary pathophysiology of back pain. *Neurosurgical Focus*. 23(1), E4, pp 1 – 6

129. Fischinger, J., A. Fischinger, and D. Fischinger, 2009. Doctor Zander's medico-mechanical institute in Opatija. *Acta Medico-Historica Adriatica*. 7(1), pp 253 – 266
130. Fisher, J., and D. Smith, 2012. Attempting to better define “intensity” for muscular performance: is it all a wasted effort? *European Journal of Applied Physiology*. 112(12), pp 4183-4185
131. Fisher, J., J. Steele, and D. Smith, 2013. Evidence based resistance training recommendations for muscular hypertrophy. *Medicina Sportiva*. 17(4), pp 217 – 235
132. Fisher, J., J. Steele, S. Bruce-Low, and D. Smith, 2011. Evidence based resistance training recommendations. *Medicina Sportiva*. 15(3), pp 147 – 162
133. Fisher, J., S. Bruce-Low, and D. Smith, 2012. A Randomized Trial to Consider the Effect of Romanian Deadlift Exercise on the development of Lumbar Extension Strength. *Physical Therapy in Sport*. 14(3), pp 139 – 145
134. Ford, D., K. M. Bagnall, K. D. McFadden, B. Greenhill, and J. Raso, 1983. Analysis of vertebral muscle obtained during surgery for correction of a lumbar disc disorder. *Acta Anatomica*. 16(2), pp 152–157
135. Fowler, N. E., Lees, A., Reilly, T., 1997. Changes in stature following plyometric drop-jump pendulum exercises. *Ergonomics*. 40(12), 1279-1286
136. Freburger, J. K., G. M. Holmes, R. P. Agans, A. M. Jackman, J. D. Darter, A. S. Wallace, L. D. Castel, W. D. Kalsbeek, and T. S. Carey, 2009. The rising prevalence of chronic low back pain. *Archives of Internal Medicine*. 169(3), pp 251 – 258
137. Frymoyer, J., 1988. Back Pain and Sciatica. *NEJM*. 318, pp 291 – 300
138. Fujiwara, A., K. Tamai, H. S. An, K. Shimizu, H. Yoshida, and K. Saotome, 2000. The interspinous ligament of the lumbar spine. Magnetic resonance images and their clinical significance. *Spine*. 25(3), pp 358 – 363

139. Fulton, M. N., 1993. *Spinal Rehabilitation (part 1) Measuring true functional ability in clinical practice*. Ocala, Florida: MedX Corporation. Available: <http://medxonline.com/downloads/articles/spinalrehabilitationpt1.pdf>
140. Garland, E. L., 2012. Pain processing in the human nervous system: a selective review of nociceptive and biobehavioural pathways. *Primary Care*. 39(3), pp 561-571
141. Gatchel, R. J., and K. H. Rollings, 2008. Evidence informed management of chronic low back pain with cognitive behavioural therapy. *The Spine Journal*. 8, pp 40 – 44
142. Gatchel, R. J., Y. B. Peng, M. L. Peters, P. N. Fuchs, and D. C. Turk, 2007. The biopsychosocial approach to chronic pain: scientific advances and future directions. *Psychological Bulletin*. 133(4), pp 581-624
143. Geisser, M. E., M. Ranavava, A. J. Haig, R. S. Roth, R. Zucker, C. Ambroz, and M. Caruso, 2005. A meta-analytic review of surface electromyography among persons with low back pain and normal, healthy controls. *The Journal of Pain*. 6(11), pp 711 – 726
144. Gibbons, L. E., T. Videman, and M. C. Batti'e, 1997. Isokinetic and psychosocial lifting strength, static back muscle endurance, and magnetic resonance imaging of the paraspinal muscles as predictors of low back pain in men. *Scandinavian Journal of Rehabilitation Medicine*. 29(3), pp 187–191
145. Gill, K., M. H. Krag, G. B. Johnson, L. D. Haugh, and M. H. Pope, 1988. Repeatability of four clinical methods for assessment of lumbar spineal motion. *Spine*. 13(1), pp 50 – 53
146. Goel, V. K., B. T. Monroe, L. G. Gilbertson, and P. Brinckman, 1995. Interlaminar shear stresses and laminae-separation in a disc: finite element analysis of the L3-L4 motion segment subjected to axial compressive loads. *Spine*. 20 (6), pp 689

147. Gonclaves, M., and F. S. S. Barbosa, 2005. Analysis of strength and resistance parameters of the lumbar spinae erector muscles during isometric exercise at different effort levels. *Rev Bras Med Esporte*. 11(2), pp 108e – 113e
148. Gracovetsky, S., 1985. An hypothesis for the role of the spine in human locomotion: A challenge to current thinking. *Journal of Biomedical Engineering*. 7, pp 205 – 216
149. Graned, H., and B. Morelli, 1988. Low back pain among retired wrestlers and heavyweight lifters. *American Journal of Sports Medicine*. 16(5), pp 530–533
150. Graves, J. E., C. K. Fix, M. L. Pollock, S. H. Leggett, D. N. Foster, and D. M. Carpenter, 1992^a. Comparison of two restraint systems for pelvic stabilisation during isometric lumbar extension strength testing. *Journal of Orthopaedic and Sports Therapy*. 15(1), p 37 – 42
151. Graves, J. E., D. C. Webb, M. L. Pollock, J. Matkozych, S. H. Leggett, D. M. Carpenter, D. N. Foster, and J. Cirulli, 1994. Pelvic stabilization during resistance training: Its effect on the development of lumbar extension strength. *Archives of Physical Medicine and Rehabilitation*. 75, pp 210 – 215
152. Graves, J. E., M. L. Pollock, A. E. Jones, A. B. Colvin, and S. H. Leggett, 1989. Specificity of limited range of motion variable resistance training. *Medicine and Science in Sport and Exercise*. 21(1), pp 84 – 89
153. Graves, J. E., M. L. Pollock, D. M. Carpenter, S. H. Leggett, A. Jones, M. MacMillan, and M. Fulton, 1990^a. Quantitative assessment of full range of motion isometric lumbar extension strength. *Spine*. 15(4), pp 289 – 294
154. Graves, J. E., M. L. Pollock, D. Foster, S. H. Leggett, D. M. Carpenter, R. Vuoso, and A. Jones, 1990^b. Effect of training frequency and specificity on isometric lumbar extension strength. *SPINE*. 15(6), pp 504 – 509

155. Graves, J. E., M. L. Pollock, S. H. Leggett, D. M. Carpenter, C. K. Fix, and M. N. Fulton, 1992^b. Limited range-of-motion lumbar extension strength training. *Medicine & Science in Sport & Exercise*. 24(1), pp 128 – 133
156. Green, H., C. Goreham, J. Ouyang, M. Ball-Burnett, and D Ranney, 1999. Regulation of fiber size, oxidative potential, and capillarization in human muscle by resistance exercise. *American Journal of Physiology*. 276, pp R591-596
157. Greenhalgh, T., and R. Peacock, 2005. Effectiveness and efficiency of search methods in systematic reviews of complex evidence: audit of primary sources. *BMJ*. 331(7524), pp 1064-1065
158. Griffith, J. F., Wang, Y. J., Antonio, G. E., Choi, K. C., Yu, A., Ahuja, A. T., Leung, P. C., 2007. Modified Pfirrmann grading system for lumbar intervertebral disc degeneration. *Spine*. 32(24), E708–E712
159. Guo, H. R., S. Tanaka, W. E. Halperin, and L. L. Cameron, 1999. Back pain prevalence in US industry and estimates of lost workdays. *American Journal of Public Health*. 89, pp 1029 – 1035
160. Hadar, H., N. Gadoth, and M. Heifetz, 1983. Fatty replacement of lower paraspinal muscles: normal and neuromuscular disorders. *American Journal of Roentgenology*. 141(5), pp 895–898
161. Hager, S. M., B. E. Udermann, D. M. Reineke, M. H. Gibson, J. M. Mayer, and S. J. Murray, 2006. Quantification of lumbar endurance on a backup lumbar extension dynamometer. *Journal of Sports Science and Medicine*. 5, pp 656 – 661
162. Haig, A. J., G. Weismann, L. D. Haugh,, M. Pope, and L. J. Grobler, 1993. Prospective evidence for change in paraspinal muscle activity after herniated nucleus pulposus. *Spine*. 18(7), pp 926
163. Hamil, J., and K. M. Kuntzen, 2009. *Biomechanical Basis of Human Movement*. 3rd ed. Philadelphia; Lippincott Williams & Wilkins

164. Hamlyn, N., D. G. Behm, and W. B. Young, 2007. Trunk muscle activation during dynamic weight training exercises and isometric instability activities. *Journal of Strength & Conditioning Research*. 21(4), pp 1108 – 1112
165. Hanada, E. Y., M. Johnson, and C. Hubley-Kozey, 2011. A comparison of trunk muscle activation amplitudes during gait in older adults with and without chronic low back pain. *PM R*. 3(10), pp 920 – 928
166. Handa, N., H. Yamamoto, T. Tani, T. Kawakami, and R. Takemasa, 2000. The effect of trunk muscle exercises in patients over 40 years of age with chronic low back pain. *Journal of Orthopaedic Science*. 5, pp 210 – 216
167. Harlick, J. C., S. Milosavljevic, and P. D. Milburn, 2007. Palpation identification of spinous processes in the lumbar spine. *Manual Therapy*. 12(1), pp 56 – 62
168. Hart, J. M., D. C. Kerrigan, J. M. Fritz, and C. D. Ingersoll, 2009. Jogging kinematics after lumbar paraspinal muscle fatigue. *Journal of Athletic Training*. 44(5), pp 475 – 481
169. Harts, C. C., P. H. Helmhout, R. A. De Bie, and J. B. Staal, 2008. A high intensity lumbar extensor strengthening program is little better than a low intensity program or a waiting list control group for chronic low back pain: a randomised clinical trial. *Australian journal of Physiotherapy*. 54, pp 23 – 31
170. Hausdorff, J. M., M. E. Nelson, D. Kaliton, J. E. Layne, M. J. Bernstein, A. Nuernberger, and M. A. F. Singh, 2001. Etiology and modification of gait instability in older adults: a randomized controlled trial of exercise. *Journal of Applied Physiology*. 90, pp 2117 – 2129
171. Hayden, J., M. W. Van Tulder, A. Malmivaara, and B. W. Koes, 2005. Exercise therapy for no-specific low back pain. *The Cochrane Database of Systematic Reviews*. 3, CD000335

172. Healey, E. L., Burden, A. M., McEwan, I. M., Fowler, N. E., 2011. Diurnal variation in stature: do those with chronic low-back pain differ from asymptomatic controls? *Clinical Biomechanics*. 26(4), 331-336
173. Healey, E. L., Fowler, N. E., Burden, A. M., McEwan, I. M., 2004. The influence of different unloading positions upon stature recovery and paraspinal muscle activity. *Clinical Biomechanics*. 20(4), 365-371
174. Helmhout, P. H., C. C. Harts, J. B. Staal, M. J. J. M. Candel, and R. A. de Bie, 2004^a. Comparison of a high intensity and a low intensity lumbar extensor training program as a minimal intervention treatment in low back pain: a randomized trial. *European Spine Journal*. 13, pp 537 – 547
175. Helmhout, P. H., C. C. Harts, W. Viechtbauer, R. A. de Bie, J. B. Staal, 2008^b. Isolated lumbar extensor strengthening versus regular physical therapy in an army working population with nonacute low back pain. *Archives of Physical Medicine and Rehabilitation*. 89(9), pp 1675 – 1685
176. Helmhout, P. H., C. Harts, J. B. Staal, and R. A. de Bie, 2004^b. Rationale and design of a multicentre randomized controlled trial on a ‘minimal intervention’ in Dutch army personnel with nonspecific low back pain. *BMC Musculoskeletal Disorders*. 5, pp 40
177. Helmhout, P. H., J. B. Staal, C. G. Maher, T. Petersen, J. Rainville, and W. S. Shaw, 2008^a. Exercise therapy and low back pain: Insights and proposals to improve the design, conduct, and reporting of clinical trials. *Spine*. 33(16), pp 1782 - 1788
178. Hepple, R. T., S. L. Mackinnon, J. M. Goodman, S. G. Thomas, and M. J. Plyley, 1997. Resistance and aerobic training in older men: effects on VO₂ peak and the capillary supply to skeletal muscle. *Journal of Applied Physiology*. 82, pp 1305-10.
179. Hepple, P., and A. R. R. Robertson, 2006. Back pain- reducing long term problems. *British Journal of General Practice*. , pp 324 – 326

180. Heydari, A., A. V. Nargol, A. P. Jones, A. R. Humphrey, and C. G. Greenough, 2010. EMG analysis of lumbar paraspinal muscles as a predictor of the risk of low-back pain. *European Spine Journal*. 19(7), pp 1145 – 1152
181. Hicks, G. E., E. M. Simonsick, T. B. Harris, A. B. Newman, D. K. Weiner, M. A. Nevitt, and F. A. Tyllavsky, 2005. Cross-sectional association between trunk muscle composition, back pain, and physical function in the health, aging and body composition study. *Journal of Gerontology*. 60(7), pp 882-887
182. Hickson, R. C., K. Hidaka, and C. Foster, 1994. Skeletal muscle fibre-type, resistance training, and strength-related performance. *Medicine and Science in Sports and Exercise*. 26, pp 593-598.
183. Hides, J. A., M. J. Stokes, M. Saide, G. A. Jull, and D. H. Cooper, 1994. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/sub-acute low back pain. *Spine*. 19, pp 165–172
184. Hides, J. A., C. A. Richardson, and G. A. Jull, 1996. Multifidus muscle recovery is not automatic after resolution of acute, first episode low back pain. *Spine*. 21(23), pp 2763 – 2769
185. Highland, T. R., and T. E. Dreisinger, 1992. Degenerative disc disease in a collegiate volleyball player. *Medicine & Science in Sport & Exercise*. 24(5), pp S163
186. Hill, A. B.. 1965. The environment and disease: Association or causation? *Proceedings of the Royal Society of Medicine* 58 (5): 295.
187. Hirsch, C., B. Ingelmark, and M. Miller, 1963. The anatomical basis for low back pain: Studies on the presence of sensory nerve endings in ligamentous, capsular and intervertebral disc structures in the human lumbar spine. *Acta Orthopaedica*. 33(1), pp 1 – 17
188. Hitselberger, W. E., and R. M. Witten, 1968. Abnormal myelograms in asymptomatic patients. *Journal of Neurosurgery*. 28, pp 204 – 206

189. Hodges, P. W., and R. J. Smeets, 2014. Interaction between pain, movement and physical activity: Short-term benefits, long-terms consequences, and targets for treatment. *Clinical Journal of Pain*. Epub ahead of print.
190. Hoeger, W. W. K., D. R. Hopkins, S. L. Barette, and D. F. Hale, 1990. Relationship between repetitions and selected percentages of one repetition maximum: a comparison between untrained and trained males and females. *Journal of Strength and Conditioning Research*. 4(2), pp 46 – 54
191. Hollingworth, W., A. K. Dixon, C. J. Todd, M. I. Bell, N. M. Antoun, Q. Arafat. S/ Girling, K. R. Karia, and R. J. Laing, 1998. Self reported health status and magnetic resonance imaging findings in patients with low back pain. *European Spine Journal*. 7, pp 369 – 375
192. Holmes, B., V. Mooney, S. Negri, S. Leggett, J. Nichols, and A. Hoeyberghs, 1996. Comparison of female geriatric lumbar extension strength: asymptomatic versus chronic low back pain patients and their response to active rehabilitation. *Journal of Spinal Disorders*. 9 (1), pp 17 – 22
193. Holmstrom, E., U. Moritz, and M. Andersson, 1992. Trunk muscle strength and back muscle endurance in construction workers with and without low back disorders. *Scandinavian Journal of Rehabilitation Medicine*. 24(1), pp 3 – 10
194. Holt, E. P., 1968. The question of lumbar discography. *Journal of Bone & Joint Surgery*. 50, pp 720 – 726
195. Honeyman, P. T., and E. A. Jacobs, 1996. Effects of culture on back pain in Australian aboriginals. *Spine*. 21(7), pp 841 – 843
196. Hultman, G. M., Nordin, H. Saraste, and H. Ohlsen, 1993. Body composition, endurance, strength, cross-sectional area, and density of MM erector spinae in men and women with and without low back pain. *Journal of Spinal Disorders*. 6(2), pp 114 – 123

197. Humphrey, A., R. A. V. Nargol, A. P. Jones, A. A. Ratcliffe, C. G. Greenough, 2005. The value of electromyography of the lumbar paraspinal muscles in discriminating between chronic-low-back-pain sufferers and normal subjects. *European Spine Journal*. 14(2), pp 175 - 184
198. Hyun, J. K., J. Y. Lee, S. J. Lee, and J. Y. Jeon, 2007. Asymmetric atrophy of multifidus muscle in patients with unilateral lumbosacral radiculopathy. *Spine*. 32(21), pp E598-E602
199. Inanami, H., 1991. Iwai Orthopedic Hospital rehabilitation program. *Paper presented at International Spinal Rehabilitation Update 1991 Symposium, Daytona*
200. International Association of the Study of Pain (IASP), 1994. *IASP Taxonomy* [online]. [19/05/2011] Available from: http://www.iasp-pain.org/AM/Template.cfm?Section=Pain_Defi...isplay.cfm&ContentID=1728
201. Iwamoto, J., H. Abe, Y. Tsukimura, and K. Wakano, 2005. Relationship between radiographic abnormalities of lumbar spine and incidence of low back pain in high school rugby players: a prospective study. *Scandinavian Journal of Medicine & Science in Sports*. 15, pp 163 – 168
202. Jensen, M. C., M. N. Brant-Zawadzki, N. Obuchowski, M. T. Modic, D. Malkasian, and J. S. Ross, 1994. Magnetic resonance imaging of the lumbar spine in people without back pain. *New England Journal of Medicine*. 331(2), pp 69 – 73
203. Johnston, B. D., 2005. The need for direct, isolated exercise in spinal strength and fitness: the effects of the medx lumbar extension machine on three case subjects. *Journal of Applied Fitness*. Pp, 1 – 9
204. Jones, A., 1993. *The lumbar spine, the cervical spine and the knee* [online]. Ocala, Florida: MedX Corporation. Available: <http://medxonline.com/downloads/SpineKnee.pdf> [accessed 15/07/2011]

205. Ju, S., G. Park, E. Kim, 2012. Effects of an exercise treatment program on lumbar extensor muscle strength and pain of rehabilitation patients recovering from lumbar disc herniation surgery. *Journal of Physical Therapy Science*. 24, pp 515-518
206. Kader, D. F., D. Wardlaw, and F. W. Smith, 2000. Correlation between the MRI changes in the lumbar multifidus muscles and leg pain. *Clinical Radiology*. 55, pp 145–149
207. Kalichman, L., P. Hodges, L. Li, A. Guermazi, and D. J. Hunter, 2010. Changes in paraspinal muscle and their association with low back pain and spinal degeneration: CT study. *European Spine Journal*. 19, pp 1136–1144
208. Kamaz, M., D. Kiresi, H. Oguz, D. Emlik, and F. Levendoglu, 2007. CT measurement of trunk muscle areas in patients with chronic low back pain. *Diagnostic and Interventional Radiology*. 13, pp 144 – 148
209. Kang, C. H., M. J. Shin, S. M. Kim, S. H. Lee, and C. S. Lee, 2007. MRI of paraspinal muscles in lumbar degenerative kyphosis patients and control patients with chronic low back pain. *Clinical Radiology*. 62(5), pp 479-486
210. Kankaanpaa, M., S Taimela, D Laaksonen, S. Hanninen, and O Airaksinen, 1998^a. Back and hip extensor fatigability in chronic low back pain patients and controls. *Archives of Physical Medicine & Rehabilitation*. 79(4), pp 412 – 417
211. Kankaanpaa, M., D. Laaksonen, S. Taimela, S. M. Kokko, O. Airaksinen, and O. Hanninen, 1998^b. Age, sex, and body mass index as determinants of back and hip extensor fatigue in the isometric Sorenson back endurance test. *Archives of Physical Medicine & Rehabilitation*. 79(9), pp 1069 – 1075
212. Kanlayanaphotporn, R., Trott, P., Williams, M., Fulton, I., 2003. Effects of chronic low back pain, age and gender on vertical spinal creep. *Ergonomics*. 46(6), 561-573

213. Kanlayanaphotporn, R., Williams, M., Fulton, I., Trott, P., 2002. Reliability of the vertical spinal creep response measured in sitting (asymptomatic and low back-pain subjects). *Ergonomics*. 45(3), 240-247
214. Katz, J. N., 2006. Lumbar disc disorders and low back pain: socioeconomic factors and consequences. *The Journal of Bone & Joint Surgery – American Volume*. 88(suppl 2), pp 21 – 24
215. Kearns, C. F., W. F. Brechue, J. Bauer, and M. L. Pollock, 1997. Muscle activation during isolated and no-isolated lumbar extension exercise. *Medicine & Science in Sport & Exercise*. 29, S165
216. Kent, P. and J. Keating, 2004. Do primary-care clinicians think that non-specific low back pain is one condition? *Spine*. 29(9), pp 1022 – 1031
217. Keyserling, W. M., G. D. Herrin, D. B. Chaffin, 1980. Isometric strength testing as a means of controlling medical incidents on strenuous jobs. *Journal of Occupational Medicine*. 22(5), pp 332–336
218. Kim, Y., J. Park, J. Hsu, K. K. Cho, Y. H. Kim, and J. K. Shim, 2010. Effects of training frequency on lumbar extension strength in patients recovering from lumbar discectomy. *Journal of Rehabilitation Medicine*. 42, pp 839 – 845
219. Kirkaldy-Willis, W. H., and C. V. Burton, 1992. *Managing Low Back Pain*. 3rd ed. New York: Churchill-Livingstone
220. Kitano, T., J. E. Zerwekh, Y. Usui, M. L. Edwards, P. L. Flicker, and V. Mooney, 1993. Biochemical changes associated with the symptomatic human intervertebral disk. *Clinical Orthopaedics and Related Research*. 293, pp 372 – 377
221. Kiter, E., T. Karaboyun, A. C. Tufan, and K. Acar, 2010. Immunohistochemical demonstration of nerve endings in iliolumbar ligament. *Spine*. 35(4), pp E101 – 104

222. Kjaer, P., T. Bendix, J. S. Sorensen, L. Korsholm, and C. Leboeuf-Yde, 2007. Are MRI-defined fat infiltrations in the multifidus muscles associated with low back pain? *BMC Medicine*. 5(2)
223. Kjaer, P., C. Leboeuf-Yde, L. Kowsholm, J. S. Sorenson, and T. Bendix, 2005. Magnetic resonance imaging and low back pain in adults: a diagnostic study of 40 year old men and women. *Spine*. 30(10), pp 1173 – 1180
224. Klein, A. B., L. Snyder-Mackler, S. H. Roy, and C. J. De Luca, 1991. Comparison of Spinal Mobility and Isometric Trunk Extensor Forces with Electromyographic Spectral Analysis in Identifying Low Back Pain. *Physical Therapy*. 71(6), pp 445–454
225. Knapik, J. J., R. H. Mawdsley, and M. U. Ramos, 1983. Angular specificity and test mode specificity of isometric and isokinetic strength training. *Journal of Orthopaedic and Sports Physical Therapy*. 5(2), pp 58 – 65
226. Kourtis, D., Magnusson, M. L., Smith, F., Hadhipavlou, A., Pope, M., 2004. Spine height and disc height changes as the effect of hyperextension using stadiometry and MRI. *Iowa Orthopaedic Journal*.. 24, 65–71
227. Kovacs, F. M., V. Abaira, A. Royuela, J. Corcol, L. Alegre, M. Tomas, M. A. Mir, A. Cano, A. Muriel, J. Zamora, M. T. G. Del Real, M. Gestoso, and N. Mufraggi, 2008. Minimum detectable and minimal clinically important changes for pain in patients with non-specific neck pain. *BMC Musculoskeletal Disorders*. 9, pp 43
228. Kraemer, W. J., K. Adams, E. Cafarelli, G. A. Dudley, C. Dooly, M. S. Feigenbaum, S. J. Fleck, B. Franklin, A. C. Fry, J. R. Hoffman, R. U. Newton, J. Potteiger, M. H. Stone, N. A. Ratamess, T. Triplett-McBride, and American College of Sports Medicine, 2002. Progression models in resistance training for healthy adults. *Medicine and Science in Sports and Exercise*. 34, pp 364-80.
229. Kraft, D. E., 2002. Low back pain in the adolescent athlete. *Paediatric Clinics of North America*. 49(3), pp 643–653

230. Kroeber, M. W., F. Unglaub, H. Wang, C. Schmid, M. Thomsen, A. Nerlich, W. Richter, 2002. New in vivo animal model to create intervertebral disc degeneration and to investigate the effects of therapeutic strategies to stimulate disc regeneration. *Spine*. 27(23), pp 2684–2690
231. Kujala, U. M., S. Taimela, T. Viljanen, H. Jutila, J. T. Viitasalo, and M. C. Battie, 1996. Physical loading and performance as predictors of back pain in healthy adults. A 5-year prospective study. *European Journal of Applied Physiology*. 73, pp 452–458
232. Ladin, Z., K. R. Murphy, and C. J. DeLuca, 1989. Mechanical recruitment of low back muscles. Theoretical predictions and experimental validation. *Spine*. 14(9), pp 927–938
233. Lamothe, C. J., A. Daffertshofer, O. G. Meijer, G. L. Moseley, P. I. Wuisman, and P. J. Beek, 2004. Effects of experimentally induced pain and fear of pain on trunk coordination and back muscle activity during walking. *Clinical Biomechanics*, 19 (6), 551 – 563
234. Lamothe, C. J., O. G. Meijer, A. D. Daffertshofer, P. I. Wuisman, and P. J. Beek, 2006^a. Effects of chronic low back pain on trunk coordination and back muscle activity during walking: changes in motor control. *European Spine Journal*. 15, pp 23 – 40
235. Lamothe, C.J., A. Daffertshofer, O. G. Meijer, and P. J Beek, 2006^b. How do persons with chronic low back pain speed up and slow down? Trunk-pelvis coordination and lumbar erector spinae activity during gait. *Gait & Posture*. 23 (2), pp 230 – 239
236. Lamothe, C. J. C., J. F. Stins, M. Pont, F. Kerckhoff, and P. J. Beek, 2008. Effects of attention on the control of locomotion in individuals with chronic low back pain. *Journal of Neuroengineering and Rehabilitation*. 25(5), pp 13

237. Lamoth, C. J., O. G. Meijer, P. I. Wuisman, J. H. van Dieen, M. F. Levin, and P. J. Beek, 2002. Pelvis-thorax coordination in the transverse plane during walking in persons with non-specific low back pain. *Spine*. 27(4), pp E92 – E99
238. Langevin, H. M., and K. J. Sherman, 2007. Pathophysiological model for chronic low back pain integrating connective tissue and nervous system mechanisms. *Medical Hypotheses*. 68(1), pp 74-80
239. Lariviere, C., M. Bilodeau, R. Forget, R. Vadeboncoeur, and H Mecheri, 2010^b. Poor back muscle endurance is related to pain catastrophizing in patients with chronic low back pain. *Spine*. 35(22), pp E1178-E1186
240. Lariviere, C., D. Gagnon, D. Gravel, and A. Bertrand-Arsenault, 2008. The assessment of back muscle capacity using intermittent static contractions. Part 1 – Validity and reliability of electromyographic indices of fatigue. *Journal of Electromyography and Kinesiology*. 18(6), pp 1006-1119
241. Lariviere, C., R. A. da Silva, A. B. Arsenault, S. Nadeau, A. Plamondon, and R. Vadenboncoeur, 2010^a. Specificity of a back muscle exercise machine in healthy and low back pain subjects. *Medicine & Science in Sports & Exercise*. 42(3), pp 592 – 599
242. Lariviere, C., R. A. da Silva, A. B. Arsenault, S. Nadeau, A. Plamondon, and R. Vadeboncoeur, 2011. Specificity of a back muscle roman chair exercise in healthy and back pain subjects. *Medicine & Science in Sport & Exercise*. 43(1), pp 157 – 164
243. Latimer, J., C. G. Maher, K. Refshauge, and I. Colaco, 1999. The reliability and validity of the Biering-Sorenson test in asymptomatic subjects and subjects reporting current or previous nonspecific low back pain. *Spine*. 24, pp 2085 -2090
244. Laurent, D., P. Portero, F. Goubel, and A. Rossi, 1993. Electromyogram spectrum changes during sustained contraction related to proton and diprotonated inorganic phosphate accumulation: a ³¹P nuclear magnetic resonance study on human calf muscles. *European Journal of Applied Physiology*. 66(3), pp 263–268

245. Lebouef-Yde, C., J. M. Lauritsen, and T. Lauritzen, 1997. Why has the search for causes of low back pain largely been nonconclusive? *Spine*. 22(8), pp 877 – 881
246. Lederman, E., 2010. The myth of core stability. *Journal of Bodywork & Movement Therapies*. 14, pp 84 – 98
247. Lee, D. G., and A. Vleeming, 1998. Impaired load transfer through pelvic girdle – a new model of altered neutral zone function. *In. Proceedings from the Third Interdisciplinary World Congress on Low Back and Pelvic Pain*. Vienna, Austria
248. Lee, D. G., and A. Vleeming, 2007. An integrated approach to the treatment of pelvic girdle pain. *In. A. Vleeming, V. Mooney, and R. Stoeckart. Movement, Stability and Lumbopelvic Pain. Integration of Research and Therapy*. New York: Churchill Livingstone, pp 621
249. Lee, J. H., Y. Hoshino, K. Nakamura, Y. Kariya, K. Saita, and K. Ito, 1999. Trunk muscle weakness as a risk factor for low back pain. A 5 year prospective study. *Spine*. 24(1), pp 54 – 57
250. Lee, K. W., J. Y. Kwon, H. S. Kim, B. S. Lee, J. Y. Kang, and W. H. Park, 2000. Back exercise program with lumbar extension resisting exercise in patients with chronic low back pain. *Journal of Korean Academy of Rehabilitation Medicine*. 24(3), PP 536 – 541
251. Leggett, S., V. Mooney, L. N. Matheson, B. Nelson, T. Dreisinger, J. V. Zytveld, and L. Vie, 1999. Restorative exercise for clinical low back pain: a prospective two-center study with 1-year follow up. *Spine*. 24(9), pp 889 – 898
252. Leino, P., S. Aro, and J. Hasan, 1987. Trunk muscle function and low back disorders: a ten year follow up study. *Journal of Chronic Diseases*. 40(4), pp 289 – 96
253. Lexell, J., and D. Downham, 1992. What is the effect of aging on type 2 muscle fibres? *Journal of Neurological Sciences*. 107(2), pp 250–251

254. Lindeberg, S., L. Cordain, and S. B. Eaton, 2003. Biological and clinical potential of a paleolithic diet. *Journal of Nutritional and Environmental Medicine*. 13(3), pp 149 – 160
255. Loeb, W. Y., 1967. Measurement of spinal posture and range of spinal movement. *Annals of Physical Medicine*. 9(3), pp 103 – 110
256. Loeser, J. D., 1980. Perspectives on pain. In: P. Turner ed. *Clinical Pharmacy and Therapeutics*. London: Macmillan
257. Long, A., R. Donelson, and T. Fung, 2004. Does it matter which exercise? A randomized control trial of exercise for low back pain. *Spine*. 29(23), pp 2593 – 2602
258. Lotz, J. C., O. K. Colliou, J. R. Chin, N. A. Duncan, and E. Lienbenberg, 1998. Compression-induced degeneration of the 16 intervertebral disc: and in vivo mouse model and finite-element study. *Spine*. 23(23), pp 2493–2506
259. Lovejoy, C. O., 2007. Evolution of the human lumbopelvic region and its relationship to some clinical deficits of the spine and pelvis. In: A. Vleeming, V. Mooney, and R. Stoeckart. *Movement, Stability and Lumbopelvic Pain. Integration of Research and Therapy*. New York: Churchill Livingstone, pp 141 – 158
260. Luoto, S., M. Heliovaara, H. Hurri, 1995. Static back endurance and the risk of low back pain. *Clinical Biomechanics*. 10(6), pp 323 – 324
261. Luthi, J. M., J. H. Howald, H. Claassen, and K. Rosler, P. Vock, and H. Hoppeler, 1986. Structural changes in skeletal muscle tissue with heavy resistance exercise. *International Journal of Sports Medicine*. 7(3), pp 123–127
262. MacDonald, D. A., G. L. Moseley, and P. W. Hodges, 2006. The lumbar multifidus: Does the evidence support clinical beliefs? *Manual Therapy*. 11, pp 254–263
263. Maclean, J. J., C. R. Lee, M. Alini, and J. C. Iatridis, 2004. Anabolic and catabolic mRNA levels of the intervertebral disc vary with the magnitude and

- frequency of in vivo dynamic compression. *Journal of Orthopaedic Research*. 22(6), pp 1193 – 1200
264. Maclean, J. J., C. R. Lee, M. Alini, and J. C. Iatridis, 2005. The effects of short-term load duration on anabolic and catabolic gene expression in the rat tail intervertebral disc. *Journal of Orthopaedic Research*. 23(5)
 265. MacRae, I. F., and V. Wright, 1969. Measurement of back movement. *Annals of the Rheumatic Diseases*. 28, pp 584
 266. Magnusson, M. L., Aleksiev, A. R., Spratt, K. F., Lakes, R. S., Pope, M. H., 1996. Hyperextension and spine height changes. *Spine*. 21(22), pp 2670 – 2675
 267. Magnusson, M. L., E. Hult, I. Lindstrom, V. Lindell, M. Pope, and T. Hansson, 1990. Measurement of time-dependent height-loss during sitting. *Clinical Biomechanics*. 5, pp 137 – 142
 268. Main, C. J., P. J. Watson, 1996. Guarded movements: development of chronicity. *Journal of Musculoskeletal Pain*. 4, pp 163–170
 269. Manchikanti, L., M. V. Boswell, V. Singh, V. Pampati, K. S. Damron, and C. D. Beyer, 2004. Prevalance of facet joint pain in chronic spinal pain of cervical, thoracic, and lumbar regions. *BMC Musculoskeletal Disorders*. 5
 270. Manchikanti, L., V. Pampati, B. Fellows, and C. E. Bakhit, 1999. Prevalance of lumbar facet joint pain in chronic low back pain. *Pain Physician*. 2(3), pp 59 – 64
 271. Maniadakis, N. And A. Gray, 2000. The economic burden of back pain in the UK. *Pain*. 84 (1), pp 95 – 103.
 272. Manniche, C., and A. Jordan, 2001^a. Letter to the editor. *Spine*. 26(7), pp 842 – 843
 273. Manniche, C., and A. Jordan, 2001^b. Letter to the editor. *Spine*. 26(8), pp 994
 274. Mannion, A. F., 1999. Fibre type characteristics and function of the human paraspinal muscles: normal values and changes in association with low back pain. *Journal of Electromyography and Kinesiology*. 9, pp 363 – 377

275. Mannion, A. F., B. R. Weber, J. Dvorak, D. Grob, and M. Muntener, 1997^a. Fibre type characteristics of the lumbar paraspinal muscles in normal healthy subjects and in patients with low back pain. *Journal of Orthopaedic Research*. 15(6), pp 881 – 887
276. Mannion, A. F., B. Connolly, K. Wood, and P. Dolan, 1997^b. The use of surface EMG power spectral analysis in the evaluation of back muscle function. *Journal of Rehabilitation Research and Development*. 34(4), pp 427 – 439
277. Mannion, A. F., G. A. Dumas, R. G. Cooper, F. J. Espinosa, M. W. Faris, and J. M Stevenson, 1997^c. Muscle fibre size and type distribution in thoracic and lumbar regions of erector spinae in healthy subjects without low back pain: normal values and sex differences. *Journal of Anatomy*. 190, pp 505 – 513
278. Mannion, A. F., L. Kaser, E. Weber, A. Rhyner, J. Dvorak, and M. Muntener, 2000. Influence of age and duration of symptoms on fibre-type distribution and size of the back muscles in chronic low back pain patients. *European Spine Journal*. 9, pp 273 – 281
279. Massey, C. D., J. Vincent, M. Maneval, and J. T. Johnson, 2005. Influence of range of motion in resistance training in women: early phase adaptations. *Journal of Strength and Conditioning Research*. 19(2), pp 409 – 411
280. Massey, C. D., J. Vincent, M. Maneval, M. Moore, and J. T. Johnson, 2004. An analysis of full range of motion vs. partial range of motion training in the development of strength in untrained men. *Journal of Strength and Conditioning Research*. 18(3), pp 518 – 521
281. Matheson, L., and V. Mooney, 2006. Employment screening and functional capacity evaluation. In: C. Liebenson. *Rehabilitation of the Spine: A Practitioners Manual*. New York: Lippincott Williams & Wilkins, pp 276

282. Matheson, L., S. Leggett, V. Mooney, K. Schneider, and J. Mayer, 2002. Contributions of aerobic fitness and back strength to lift capacity. *Spine*. 27(11), pp 1208 – 1212
283. Mattila, M., M. Hurme, H. Alaranta, L. Paljarvi, H. Kalimo, B. Falck, M. Lehto, S. Einola, and M. Jarvinen, 1986. The multifidus muscle in patients with lumbar disc herniation. A histochemical and morphometric analysis of intraoperative biopsies. *Spine*. 11(7), pp 737–738
284. Maurer, M., R. B. Soder, and M. Baldisserotto, 2011. Spine abnormalities depicted by magnetic resonance imaging in adolescent rowers. *American Journal of Sports Medicine*. 39(2), pp 392 – 397
285. Mayer, J. M., B. E. Udermann, J. E. Graves, and L. L. Ploutz-Snyder, 2003. Effect of roman chair exercise training on the development of lumbar extension strength. *Journal of Strength & Conditioning Research*. 17(2), pp 356 – 361
286. Mayer, J. M., J. E. Graves, B. E. Udermann, and L. L. Ploutz-Snyder, 2002^a. Development of lumbar extension strength: Effect of pelvic stabilization during resistance training. *Journal of Back and Musculoskeletal Rehabilitation*. 16, pp 25 – 31
287. Mayer, J. M., J. E. Graves, V. L. Robertson, E. A. Pierra, J L. Verna, and L. L. Ploutz-Snyder, 1999. Electromyographic activity of the lumbar extensor muscles: Effect of angle and hand position roman chair exercise. *Archives of Physical Medicine and Rehabilitation*. 80, pp 751 – 755
288. Mayer, J. M., J. L. Verna, T. M. Manini, V. Mooney, and J. E. Graves, 2002^b. Electromyographic activity of the trunk extensor muscles: effect of varying hip position and lumbar posture during roman chair exercise. *Archives of Physical Medicine and Rehabilitation*. 83(11), pp 1453 – 1546

289. Mayer, J., V. Mooney, and S. Dagenais, 2008. Evidence informed management of chronic low back pain with lumbar extensor strengthening exercises. *The Spine Journal*. 8, pp 96 – 113
290. Mayer, T. G., G. Kondraske, V. Mooney, T. W. Carmichael, and R. Butsch, 1989^a. Lumbar myoelectric spectral analysis for endurance assessment. A comparison of normal and deconditioned patients. *Spine*. 14(9), pp 989 – 991
291. Mayer, T. G., H. Vanharanta, R. J. Gatchel, V. Mooney, D. Barnes, L. Judge, S. Smith, and A. Terry, 1989^b. Comparison of CT scan muscle measurements and isokinetic trunk strength in postoperative patients. *Spine*. 14(1), pp 33 – 36
292. Mayer, T., R. Gatchel, J. Betancur, and E. Bovasso, 1995. Trunk muscle endurance measurement: isometric contrasted to isokinetic testing in normal subjects. *Spine*. 20, pp 920 – 927
293. Mazzetti, S. A., W. J. Kraemer, J. S. Volek, N. D. Duncan, N. A. Ratamess, A. L. Gomez, R. U. Newton, K. Hakkinen, and S. J. Fleck, 2000. The Influence of direct supervision of resistance training on strength performance. *Medicine and Science in Sports and Exercise*. 32, pp 1175-84.
294. McCarron, R. F., M. W. Wimpee, P. G. Hudkins, and G. S. Laros, 1987. The inflammatory effect of nucleus pulposus. A possible element in the pathogenesis of low back pain. *Spine*. 12(8), pp 760 – 764
295. McGill, S., D. Juker, and P. Kropf, 1996. Appropriately places surface EMG electrodes reflect deep muscle activity (psoas, quadratus lumborum, abdominal wall) in the lumbar spine. *Spine*. 29(11), pp 1503-1507
296. McGill, S. M., 2007. *Low back disorders: evidence-based rehabilitation and prevention*. 2nd ed. Human Kinetics: Champaign
297. McGill, S. M., A. Childs, and C. Liebenson, 1999. Endurance times for low back stabilization exercise: clinical targets for testing and training from a normal database. *Archives of Physical Medicine and Rehabilitation*. 80, pp 941 – 944

298. McGill, S. M., and V. R. Yingling, 1999. Traction may enhance the imaging of the spine versus plane radiographs: implications for the laboratory versus the clinic. *Clinical Biomechanics*. 14(4), pp 291 – 295
299. McGill, S. M., van Wijk, M. J., Axler, C. T., Gletsu, M., 1996. Studies of spinal shrinkage to evaluate low-back loading in the workplace. *Ergonomics*. 39(1), 92-102
300. McMahon, G E., C. I. Morse, A. Burden, K. Winwood, and G. L. Onambélé, 2014. Impact of range of motion during ecologically valid resistance training protocols, on muscle size, subcutaneous fat and strength. *J Strength Cond Res*. 28(1), pp 245 – 255
301. McNee, P., J. Shambrook, E. C. Harris, M. Kim, M. Sampson, K. T. Palmer, and D. Coggon, 2011. Predictors of long term pain and disability in patients with low back pain investigated by magnetic resonance imaging: A longitudinal study. *BMC Musculoskeletal Disorders*. 12, pp 234
302. McNeill, T., D. Warwick, G. Andersson, and A. Schultz, 1980. Trunk strengths in attempted flexion, extension, and lateral bending in healthy subjects and patients with low back disorders. *Spine*. 5(6), pp 529 – 538
303. MedX Corporation, 2008. *Facility Locator* [online]. Available: <http://medxonline.com/locator.php> [accessed 16/01/2009]
304. MedX Utilisation Steering Committee, 1995-1996. *Consensus guidelines for the utilization of MedX medical testing and exercise machines in spinal rehabilitation programs*. Ocala, Florida: MedX Corporation.
305. Mengiardi, B., Am. R. Schmid, N. Boos, C. W. A. Pfirrmann, F. Brunner, A. Elfering, and J. Hodler, 2006. Fat content of lumbar paraspinal muscles in patients with chronic low back pain and asymptomatic volunteers: Quantification with MR spectroscopy. *Radiology*. 240(3), pp 786 – 792

306. Miltner, O., D. C. Wirtz, and C. H. Siebert, 2001. Lumbar extension exercise (MedX) – Treatment for chronic low back pain – Review and Meta-Analysis. *Zeitschrift fur Orthopadie und Unfallchirurgie*. 139(4), pp 287–293
307. Mimura, M., M. M. Panjabi, T. R. Oxland, J. J. Crisco, I. Yamamoto, and A. Vasavada, 1994. Disc degeneration affects the multidirectional flexibility of the lumbar spine. *Spine*. 19(12), pp 1371-1380
308. Moffroid, M. T., L. D. Haugh, A. J. Haig, S. M. Henry, and M. H. Pope, 1993. Endurance training of trunk extensor muscles. *Physical Therapy*. 73(1), pp 10 – 17
309. Moffroid, M., S. Reid, S. M. Henry, L. D. Haugh, and A. Ricamato, 1994. Some endurance measures in persons with chronic low back pain. *Journal of Orthopedic and Sports Physical Therapy*. 20(2), pp 81 – 87
310. Mooney, V., K. Kenney, S. Leggett, and B. Holmes. Relationship of lumbar strength in shipyard workers to workplace injury claims. *Spine*. 21(17), pp 2001–2005
311. Mooney, V. K. Kenney, S. Leggett, and B. Holmes, 1996. Relationship of lumbar strength in shipyard workers to workplace injury claims. *Spine*. 21(17), pp 2001 – 2005
312. Mooney, V., 1992. On the dose of therapeutic exercise. *Orthopedics*. 15(5), pp 653 – 656
313. Mooney, V., 2007. *The Unguarded Moment: A Surgeon's Discovery of the Barriers to Prescription of Inexpensive, Effective Healthcare in the form of Therapeutic Exercise*. Vantage Press: New York
314. Mooney, V., and G. B. J. Andersson, 1994. Controversies. Trunk strength testing in patient evaluation and treatment. *Spine*. 19(21), pp 2483 – 2485
315. Mooney, V., J. Gulick, M. Perlman, D. Levy, R. Pozos, S. Leggett, and D. Resnick, 1997. Relationships between myoelectric activity, strength and MRI of lumbar extensor muscles in back pain patients and normal subjects. *Journal of Spinal Disorders*. 10(4), pp 348 – 356

316. Mooney, V., J. Verna, and C. Morris, 2006. Clinical management of chronic, disabling low back syndromes. In. C. Morris, ed. *Rehabilitation of the Spine: A Practitioners Manual*. New York: McGraw-Hill
317. Mooney, V., L. Matheson, D. Holmes, S. Leggett, J. Grant, S. Negri, and B. Holmes, 1993. Effect of focused strength training after low back injury. *Paper presented at North American Spine Society 1993 Annual Meeting, San Diego, California*
318. Mooney, V., M. Kron, P. Rummerfield, and B. Holmes, 1995. The effect of workplace based strengthening on low back injury rates: A case study in the strip mining industry. *Journal of Occupational Rehabilitation*. 5(3), pp 157 – 167
319. Mooney, V., R. Pozos, A. Vleeming, J. Gluick, and D. Swenski, 2001. Exercise treatment for sacroiliac pain. *Orthopedics*. 24(1), pp 29 – 32
320. Mootz, R. D., J. C. Keating Jr, H. P. Kontz, T. B. Milus, and G. E. Jacobs, 1989. Intra- and interobserver reliability of passive motion palpation of the lumbar spine. *Journal of Manipulative and Physiological Therapies*. 12(6), pp 440 – 445
321. Mori, A., 2004. Electromyographic activity of selected trunk muscles during stabilization exercises using a gym ball. *Electromyographic and Clinical Neurophysiology*. 4(1), pp 57 – 64
322. Moseley, G. L., P. W. Hodges, and S. C. Gandevia, 2002. Deep and superficial fibers of the multifidus are differentially active during voluntary arm movements. *Spine*. 27(2), pp E29–36
323. Mostardi, R. A., D. A. Noe, M. W. Kovacik, and J. A. Porterfield, 1992. Isokinetic lifting strength and occupational injury. A prospective study. *Spine*. 17(2), pp 189–193
324. Motosuneya, T., T. Asazume, T. Tsuji, H. Watanabe, Y. Nakayama, and K. Nemoto, 2006. Post-operative change of the cross-sectional area of back

- musculature after 5 surgical procedures as assessed by magnetic resonance imaging. *Journal of Spinal Disorders and Techniques*. 19(5), pp 318–322
325. Murray, D., 1986. Optimal filtering of constant velocity torque data. *Medicine & Science in Sport & Exercise*. 18, pp 603 – 611
 326. Nachemson, A. L., G. Waddell, and A. I. Norlund, 2000. Epidemiology of low back pain. In: A. L. Nachemson, and E. Jonsson (eds). *Neck and back pain: The scientific evidence of causes, diagnosis and treatment*. Philadelphia: Lippincott Williams & Wilkins
 327. National Institute for Health and Clinical Excellence (NICE), 2009. *Low back pain: early management of persistent non-specific low back pain*. London: Royal College of General Practitioners
 328. National Research Council (NRC), 1998. *Work-related musculoskeletal disorders: A Review of the evidence*. National Academy Press: Washington, DC
 329. National Research Council (NRC), and The Institute of Medicine (IOM), 2001. *Musculoskeletal disorders and the workplace: Low back and upper extremities*. National Academy Press: Washington, DC
 330. Nayha, S., T. Videman, M. Laakso, and J. Hassi, 1991. Prevalence of Low Back Pain and Other Musculoskeletal Symptoms and Their Association with Work in Finnish Reindeer Herders. *Scandinavian Journal of Rheumatology*. 20(6), pp406 - 413
 331. Nelson, B. W., D. M. Carpenter, T. E. Dreisinger, M. Mitchell, C. E. Kelly, and J. A. Wegner, 1999. Can spinal surgery be prevented by aggressive strengthening exercise? A prospective study of cervical and lumbar patients. *Archives of Physical Medicine and Rehabilitation*. 80, pp 20 – 25
 332. Nelson, B. W., E. O'Reilly, and M. Miller, 1995. The clinical effects of intensive, specific exercise on low back pain: A controlled study of 895 consecutive patients with a one year follow up. *Orthopedics*. 18 (10), pp 971 – 981.

333. Newton, M., M. Thow, D. Somerville, I. Henderson, and G. Waddell, 1993. Trunk strength testing with iso-machines. Part 2. Experimental evaluation of the cybex II back testing system in normal subjects and patients with chronic low back pain. *Spine*. 18(7), pp 812 – 824
334. Nicholaisen, T., and K. Jorgensen, 1985. Trunk muscle strength, back muscle endurance and low back trouble. *Scandinavian Journal of Rehabilitation Medicine*. 17(3), pp 121 – 127
335. Nicodemus, C. L., 1999. Biomechanical study on the efficacy of back strengthening methods and machines. *Proceedings of the American Back Society*. Dec 8-11, Las Vegas, Nevada
336. Norris, C. M., 2008. *Back Stability: Integrating science and therapy*. Champaign: Human Kinetics
337. O'Dwyer, N., R. Smith, M. Kalaki, and U. Rattanaprasert, 2009. Independent assessment of pattern and offset variability of time series waveforms. *Gait & Posture*. 29, pp 285 – 289
338. Office for National Statistics (ONS), 1997. *The Health of Adult Britain: 1841 – 1994. Volume 2*. London: The Stationary Office
339. Office for National Statistics (ONS), 2000. *Social Trends 30*. London: The Stationary Office
340. Office for National Statistics (ONS), 2008. *All items Retail Price Index (RPI) percentage change over 12 months: Table RP04*. [online] Available: <http://www.statistics.gov.uk/default.asp> [accessed 24/07/2009]
341. Office for National Statistics (ONS), 2010. *Social Trends 40*. Basingstoke: Palgrave Macmillan
342. Ogon, M., M. Krismer, W. Sollner, W. Kantner-Rumplmair, and A. Lampe, 1996. Chronic low back pain measurement with visual analogue scales in different settings. *Pain*. 64(3), pp 425 – 428

343. Ostelo, R. W. J. G., R. A. Deyo, P. Stratford, G. Waddell, P. Croft, M. Von Korf, L. M. Bouter, and H. C. De Vet, 2008. Interpreting change scores for pain and functional status in low back pain: Towards international consensus regarding minimal important change. *Spine*. 33(1), pp 90 – 94
344. Owens, S. C., Brismee, J., Penell, P. N., Dedrick, G. S., Sizer, P. S., James, C. R., 2009. Changes in spinal height following sustained lumbar flexion and extension postures: A clinical measure of intervertebral disc hydration using stadiometry. *Journal of Manipulative and Physiological Therapeutics*. 32(5), 358-363
345. Paajanen, H., Lehto, I., Alanen, A., Erkinntalo, M., Komu, M., 1994. Diurnal fluid changes of lumbar discs measured indirectly by magnetic resonance imaging. *Journal of Orthopaedic Research*. 12(4), 509-514
346. Paasuke, M., E. Johanson, M. Proosa, J. Ereline, and H. Gapeyeva, 2002. Back extensor fatigability in chronic low back pain patients and controls: Relationship between electromyogram power spectrum changes and body mass index. *Journal of Back and Musculoskeletal Rehabilitation*. 16(1), pp 17 – 24
347. Panjabi, M., K. Abumi, J. Duranceau, and T. Oxland, 1989. Spinal stability and intersegmental muscle forces. A biomechanical model. *Spine*. 14(2), pp 194–200
348. Panjabi, M. M., 2006. A hypothesis of chronic back pain: ligament subfailure injuries lead to muscle control dysfunction. *European Spine Journal*. 15, pp 668 – 676
349. Papadakis, N. C., D. G. Christakis, G. N. Tzagarakis, G. I. Chlouverakis, N. A. Kampanis, K. N. Stergiopoulos, and P. G. Katonis, 2009. Gait variability measurements in lumbar spinal stenosis patients: part A comparison with healthy subjects. *Physiological Measurement*. 30 (11), pp 1171 - 1186
350. Papageorgiou, A. C., P. R. Croft, E. Thomas, S. Ferry, M. I. Jayson, and A. J. Silman, 1996. Influence of previous pain experience on the episode incidence of low

back pain: results from the South Manchester Back Pain study. *Pain*, 66(2-3), pp 181 – 185

351. Park, Y. J., K. S. Choi, and S. G. Lee, 2000. Effect of lumbar extensor strengthening in chronic low back pain patients. *Journal of Korean Academy of Rehabilitation Medicine*. 24(2), pp 295 – 300
352. Parkkola, R., U. Kujala, and U. Rytokoski, 1992. Response of the trunk muscles to training assessed by magnetic resonance imaging and muscle strength. *European Journal of Applied Physiology & Occupational Physiology*. 65(5), pp 383 – 387
353. Parkkola, R., U. Rytokoski, and M. Kormano, 1993. Magnetic resonance imaging of the discs and trunk muscles in patients with chronic low back pain and healthy control subjects. *Spine*. 18(7), pp 830 – 836
354. Pauly, J. E., 1966. An electromyographic analysis of certain movements and exercises. I. some deep muscles of the back. *The Anatomical Record*. 155(2), pp 223 – 234
355. Peach, J. P., and S. M. McGill, 1998. Classification of low back pain with the use of spectral electromyogram parameters. *Spine*. 23(10), pp 1117 – 1123
356. Percy, M., I. Portek, and J. Shepherd, 1985. The effect of low back pain on lumbar spinal movements measured by three-dimensional x-ray analysis. *Spine*. 10(2), pp 150 – 153
357. Perez, L. T., O. B. Peiro, T. P. Dies, R. M. Navas, P. G. Ballarini, and M. B. Vives, 2007. Fuerza lumbar en jugadores de hockey hierba. *Apunts. Medicina De L'Esport*. 155, pp 138-144
358. Petersen, C. M., L. R. Amundsen, and M. J. Schendel, 1987. Comparison of the effectiveness of two pelvic stabilization systems on pelvic movement during maximal isometric trunk extension and flexion muscle contractions. *Physical Therapy*. 67(4), pp 534 – 539

359. Pinto, R. S., N. Gomes, R. Radaelli, C. E. Botton, L. E. Brown, and M. Bottaro, 2012. Effects of range of motion on muscle strength and thickness. *Journal of Strength and Conditioning Research*. 26(8), pp 2140 – 2145
360. Pitcher, M. J., D. G. Behm, and S. N. MacKinnon, 2008. Reliability of electromyographic and force measures during isometric back extension in subjects with and without low back pain. *Applied Physiology, Nutrition and Metabolism*. 33(1), pp 52-60
361. Plowman, S. A., 2001. Muscular strength, endurance and flexibility. <http://www.cooperinst.org/ftgrefintro.asp>. Cited In: N. S. Hannibal III, S. A. Plowman, M. A. Looney, and J. Brandenburg. Reliability and vaildty of low back strength/muscular endurance field tests in adolescents. *Journal of Physical Activity and Health*. 3(Supp. 2), pp 78 – 79
362. Pollock, M. L., J. E. Graves, D. M. Carpenter, D. Foster, S. H. Leggett, and M. N. Fulton, 1993. Muscle. In: S. H. Hochschuler, H. B. Cotler, and R. D. Guyer. *Rehabilitation of the Spine: Science and Practice*. St. Louis: Mosby, pp 263 – 284
363. Pollock, M. L., J. E. Graves, S. H. Leggett, et al. 1991. Accuracy of counter weighting to account for upper body mass in testing lumbar extension strength. *Medicine & Science in Sport & Exercise*. 23, pp 66
364. Pollock, M. L., S. H. Leggett, J. E. Graves, A. Jones, M. Fulton, and J. Cirulli, 1989. Effect of resistance training on lumbar extension strength. *The American Journal of Sports Medicine*. 17(5), pp 624 – 629
365. Price, D. D., 2000. Psychological and neural mechanisms of the affective dimension of pain. *Science*. 288, pp 1769 – 1772
366. Price, W. A., 2008. *Nutrition and Physical Degeneration*. 8th ed. California: Price-Pottenger Foundation

367. Punnett, L., and D. H. Wegnam, 2004. Work-related musculoskeletal disorders: the epidemiological evidence and the debate. *Journal of Electromyography and Kinesiology*. 14, pp 13 – 23
368. Rantanen, J., M. Hurme, B. Falck, H. Alaranta, F. Nykvist, M. Lehto, S. Einola, and H. Kalimo, 1993. The lumbar multifidus muscle five years after surgery for lumbar intervertebral disc herniation. *Spine*. 18(5), pp 568–574
369. Reilly, T., Tyrell, A., Troup, J. D., 1984. Circadian variation in human stature. *Chronobiol. Int.* 1(2), 121-126
370. Reimer, D. S., B. D. Halbrook, P. H. Dreyfuss, and C. Tibletti, 1994. A novel approach to preemployment worker fitness evaluations in a material handling industry. *Spine*. 19(18), pp 2026–2032
371. Renkawitz, T., D. Boluki, and J. Grifka, 2006. The association of low back pain, neuromuscular imbalance and trunk extension strength in athletes. *The Spine Journal*. 6, pp 673-683
372. Reyna, J. R., S. H. Leggett, K. Kenney, B. Holmes, and V. Mooney, 1995. The effect of lumbar belts on isolated lumbar muscle. Strength and dynamic capacity. *Spine*. 20(1), pp 68 – 73
373. Ricci, J. A., W. F. Stewart, E. Chee, C. Leotta, K. Foley, and M. C. Hochberg, 2006. Back pain exacerbations and lost productive time costs in United States workers. *Spine*. 31(26), pp 3052 – 3060
374. Richmond, J., 2012. Multi-factorial causative model for back pain management; relating causative factors and mechanisms to injury presentations and designing time- and cost effective treatment thereof. *Medical Hypotheses* 79(2), pp 232-240

375. Risch, S. V., N. K. Norvell, M. L. Pollock, E. D. Risch, H. Langer, M. Fulton, J. E. Graves, and S. H. Leggett, 1993. Lumbar strengthening in chronic low back pain patients. *Spine*. 18(2), pp 232 – 238
376. Rissanen, A., M. Heliovaara, H. Alaranta, S. Taimela, E. Malkia, P. Knekt, A. Reunanen, and A. Aromaa, 2002. Does good trunk extensor performance protect against back-related work disability. *Journal of Rehabilitation Medicine*. 34, pp 62-66
377. Roberts, T. J., and A. M. Gabaldon, 2008. Interpreting muscle function from EMG: lessons learned from direct measurements of muscle force. *Integrative and Comparative Biology*. 48(2), pp 312–320
378. Robinson, M. E., A. F. Greene, P. O'Connor, J. E. Graves, and M. MacMillan, 1992^a. Reliability of lumbar isometric torque in patients with chronic low back pain. *Physical Therapy*. 72(3), pp 186 – 190
379. Robinson, M. E., J. E. Cassisi, P. D. O'Connor, M. MacMillan, 1992^b. Lumbar iEMG during isotonic exercise: chronic low back pain patients vs. controls. *Journal of Spinal Disorders*. 5(1), pp 8 – 15
380. Rodacki, A. L. F., Weidle, C. M., Fowler, N. E., Rodacki, C. L. N., Persch, L. N., 2007. Changes in stature during and after spinal traction in young male subjects. *Rev. Bras. Fisioter. Sao. Carlos*. 11(1), 63-71
381. Rodacki, C. L., Fowler, N. E., Rodacki, A. L., Birch, K., 2001. Technical note: repeatability of measurement in determining stature in sitting and standing postures. *Ergonomics*. 44(12), 1076-1085
382. Rodacki, C. L., Fowler, N. E., Rodacki, A. L., Birch, K., 2003. Stature loss and recovery in pregnant women with and without low back pain. *Archives of Physical Medicine and Rehabilitation*. 84(4), 507-512

383. Rodney, K. J., R. D. Herbert and R. J. Balnave, 1994. Fatigue contributes to the strength training stimulus. *Medicine & Science in Sport & Exercise*. 26, pp 1160 – 1164
384. Roman-Liu, D., and T. Tokarski, 2002. EMG of arm forearm muscle activities with regard to handgrip force in relation to upper limb location. *Acta Bioengineering and Biomechanics*. 4(2), pp 33–48
385. Rosatelli, A. L., K. Ravichandrian, and A. M. Agur, 2008. Three-dimensional study of the musculotendinous architecture of the lumbar multifidus and its functional implications. *Clinical Anatomy*. 21, pp 539–546
386. Roy, S. H., C. J. De Luca, D. A. Casavant, 1989. Lumbar muscle fatigue and chronic low back pain. *Spine*. 14(9), pp 992 – 1001
387. Roy, S. H., C. J. De Luca, L. Snyder-Mackler, M. S. Emley, R. L. Crenshaw, and J. P. Lyons, 1990. Fatigue, recovery, and low back pain in varsity rowers. *Medicine & Science in Sport & Exercise*. 22(4), pp 463 – 469
388. Roy, S. H., C. J. De Luca, M. Emley, and R. J. C. Buijs, 1995. Spectral electromyographic assessment of back muscles in patients with low back pain undergoing rehabilitation. *Spine*.(1), pp 38 – 48
389. Saal, J. S., R. C. Franson, R. Dobrow, J. A. Saal, A. H. White, and N. Goldthwaite, 1990. High levels of inflammatory phospholipase A2 activity in lumbar disc herniations. *Spine*. 15(7), pp 674 – 678
390. Salminen, J. J., M. Erkinntalo, M. Laine, and J. Pentti, 1995. Low back pain in the young. A prospective three year follow up study of subjects with and without low back pain. *Spine*. 20(19), pp 2101 – 2107
391. Salminen, J. J., P. Maki, A. Okansen, and J. Pentti, 1992. Spinal mobility and trunk muscle strength in 15 year old school children with and without low back pain. *Spine*. 17(4), pp 405 – 411

392. Salzberg, L., 2012. The physiology of low back pain. *Primary Care*. 39(3), pp 487-498
393. San Juan, J. G., J. A. Yaggie, S. Levy, V. Mooney, B. Udermann, and J. M. Mayer, 2005. Effects of pelvic stabilisation on lumbar muscle activity during dynamic exercise. *Journal of Strength and Conditioning Research*. 19(4), pp 77 – 81
394. Schache, A. G., P. Blanch, D. Rath, T. Wrogley, and K. Bennell, 2002^a. Three-dimensional angular kinematics of the lumbar spine and pelvis during running. *Human Movement Science*. 21, pp 273 – 293
395. Schache, A. G., P. D. Blanch, D. A. Rath, T. V. Wrigley, R. Starr and K. L. Bennell, 2002^b. Intra-subject repeatability of the three dimensional angular kinematics within the lumbo-pelvic-hip complex during running. *Gait & Posture*. 15, pp 136 – 145
396. Schmidt-Wilcke, T., E. Leinisch, S. Ganssbauer, B. Draganski, U. Bogdahn, J. Altmepfen, and A. May, 2006. Affective component and intensity of pain correlate with structural differences in gray matter in chronic back pain patients. *Pain*. 125(1-2), pp 89 – 97
397. Schober, P., 1937. The lumbar vertebral column and backache. *Munch Med Wschr*. 84, pp 336
398. Schott, J., K. McCully, and O. M. Rutherford, 1995. The role of metabolites in strength training. Short versus long isometric contractions. *European Journal of Applied Physiology*. 71, pp 337 – 341
399. Seay, J. F., R. E. Van Emmerik, and J. Hamill, 2011^a. Influence of low back pain status on pelvis-trunk coordination during walking and running. *Spine*. 36(16), pp E1070 – E1079
400. Seay, J. F., R. E. Van Emmerik, and J. Hamill, 2011^b. Low back pain status affects pelvis-trunk coordination and variability during walking and running. *Clinical Biomechanics*. 26(6), pp 572 – 578

401. Semmler, J. G., K. J. Tucker, T. J. Allen, and U. Proske, 2007. Eccentric exercise increases EMG amplitude and force fluctuations during submaximal contractions of elbow flexor muscles. *Journal of Applied Physiology*. 103, pp 979–989
402. Shambrook, J., P. McNee, E. C. Hariis, M. Kim, M. Sampson, K. T. Palmer, and D. Coggon, 2011. Clinical presentation of low back pain and association with risk factors according to findings on magnetic resonance imaging. *Pain*. 152, pp 1659 – 1665
403. Shimano, T., W. J. Kraemer, B. A. Spiering, J. S. Volek, D. L. Hatfield, R. Silvestre, J. L. Vingren, M. S. Fragala, C. M. Maresh, S. J. Fleck, R. U. Newton, L. P. Spreuwenberg, and K. Hakkinen, 2006. Relationship between the number of repetitions and selected percentages of one repetition maximum in free weight exercises in trained and untrained men. *Journal of Strength and Conditioning Research*. 20(4), pp 819 – 823
404. Shirado, O., T. Ito, K. Kaneda, and T. E. Strax, 1995. Electromyographic analysis of four techniques for isometric trunk muscle exercises. *Archives of Physical Medicine and Rehabilitation*. 76, pp 225 – 229
405. Shirley, F. R., P. O'Connor, M. E. Robinson, and M. MacMillan, 1994. comparison of lumbar range of motion using three measurement devices in patients with chronic low back pain. *Spine*. 19, pp 779 – 793
406. Siddall, P. J., and M. J. Cousins, 1997. Spinal pain mechanisms. *Spine*. 22(1), pp 98 – 104
407. Sihvonen, T., A. Herno, L. Paljarvi, O. Airaksinen, J. Pantanen, and A. Tapaninaho, 1993. Local denervation atrophy of paraspinal muscle in postoperative failed back syndrome. *Spine*. 18(5), pp 575–581
408. Sirca, A., and V. Kostevc, 1985. The fibre type composition of thoracic and lumbar paravertebral muscles in man. *Journal of Anatomy*. 141, pp 131–137

409. Sjolie, A. N., and A. E. Ljunggren, 2001. The significance of high lumbar mobility and low lumbar strength for current and future low back pain in adolescents. *Spine*. 26(23), pp 2629 – 2636
410. Slade, S. C., and J. L. Keating, 2006. Trunk-strengthening exercise for chronic low back pain: A systematic review. *Journal of Manipulative and Physiological Therapeutics*. 29(2), pp 163 – 173
411. Smeets, R. J. E. M., D. Wade, A. Hidding, P. J. C. M. Van Leeuwen, J. W. S. Vlaeyen, and J. A. Knottnerus, 2006. The association of physical deconditioning and chronic low back pain: A hypothesis oriented systematic review. *Disability & Rehabilitation*. 28(11), pp 673 – 693
412. Smidt, G., T. Herring, L. Amundsen, M. Rogers, A. Russell, and T. Lehmann, 1983. Assessment of abdominal and back extensor function. *Spine*. 8(2), pp 211 – 219
413. Smith, D., G. Bissell, S. Bruce-Low, and C. Wakefield, 2011. The effect of lumbar extension training with and without pelvic stabilization on lumbar strength and low back pain. *Journal of Back and Musculoskeletal Rehabilitation*. 24, pp 1 – 9
414. Smith, D., S. Bruce-Low, and G. Bissell, 2008. Twenty years of specific, isolated lumbar extension research: A review. *Journal of Orthopaedics*. 5 (1), e14
415. Solomonow, M., B. H. Zhou, M. Harriz, Y. Lu, and R. V. Baratta, 1998. The ligamento-muscular stabilizing system of the spine. *Spine*. 23(23), pp 2552–2562
416. Souza, G. M., L. L. Baker, and C. M. Powers, 2001. Electromyographic activity of selected trunk muscles during dynamic spine stabilization exercises. *Archives of Physical Medicine and Rehabilitation*. 82, pp 1551 – 1557
417. Sparto, P. J., M. Parniapour, T. E. Reinsel, and S. Simon, 1997^b. The effect of fatigue of multijoint kinematics and load sharing during a repetitive test. *Spine*. 22(22), pp 2647 – 2654

418. Sparto, P. J., M. Parniapor, W. S. Marras, K. P. Granata, T. E. Reinsel, and S. Simon, 1997^a. Neuromuscular trunk performance and spinal loading during a fatiguing isometric trunk extension with varying torques requirements. *Journal of Spinal Disorders*. 10(2), pp 145 – 156
419. Steele, J., S. Bruce-Low, D. Smith, D. Jessop, and N. Osborne, 2013^a. A Randomised Controlled Trial of Limited Range of Motion Lumbar Extension Exercise in Chronic Low Back Pain. *Spine*. 38(15), pp 1245-1252
420. Steele, J., S. Bruce-Low, and D. Smith, 2014^a. A reappraisal of the deconditioning hypothesis in low back pain: review of evidence from a triumvirate of research methods on specific lumbar extensor deconditioning. *Current Medical Research and Opinion*. 30(5), pp 865-911
421. Steele, J., S. Bruce-Low, and D. Smith, 2013^c. A review of the specificity of exercises designed for conditioning the lumbar extensors. *British Journal of Sports Medicine*. Online First
422. Steele, J., J. Fisher, D. McGuff, S. Bruce-Low, and D. Smith, 2012. Resistance training to momentary muscular failure improves cardiovascular fitness in humans: A review of acute physiological responses and chronic physiological adaptations. *Journal of Exercise Physiology*. 15(3), pp 53–80
423. Steele, J., 2013^a. An ancient perspective on deconditioning in low back pain. Presented at *Ancestral Health Symposium*, Atlanta Sheraton Hotel
424. Steele, J., 2013^b. Intensity; in-ten-si-ty; noun. 1. Often used ambiguously within resistance training. 2. Is it time to drop the term altogether? *British Journal of Sports Medicine*. Online First
425. Steele, J., and S. Bruce-Low, 2012. Steiger et al. 2011: relationships and specificity in CLBP rehabilitation through exercise. *European Spine Journal*. DOI: 10.1007/s00586-012-2449-y Online First

426. Steele, J., S. Bruce-Low, and D. Smith, 2013^b. Controlling Resistance Training Variables in Interventions for Chronic Nonspecific Low Back Pain: Letter to the Editor regarding “The Effects of Dynamic Isolated Lumbar Extensor Training on Lumbar Multifidus Functional Cross-Sectional Area and Functional Status of Patients with Chronic Nonspecific Low Back Pain”. *Spine*. 38(18), pp 1609 – 1610
427. Steele, J., S. Bruce-Low, D. Smith, N. Osborne, and A. Thorkeldsen, 2014^b. Can specific loading through exercise impart healing or regeneration of the intervertebral disc? *The Spine Journal*. In Press
428. Steering Committee for the Review of Government Service Provision, 2007. *Overcoming Indigenous Disadvantage: Key Indicators 2007*. Productivity Commission: Canberra
429. Steiger, F., B. Wirth, E. D. de Bruin, and A. F. Mannion, 2012^a. Is a positive clinical outcome after exercise therapy for chronic non-specific low back pain contingent upon a corresponding improvement in targeted aspects(s) of performance? A systematic review. *European Spine Journal*. 21, pp 575 – 598
430. Steiger, F., B. Wirth, E. D. de Bruin, and A. F. Mannion, 2012^b. Answer to the Letter to the Editor of J. Steele et al. concerning manuscript “Is a positive clinical outcome after exercise therapy for chronic non-specific low back pain contingent upon a corresponding improvement in targeted aspects(s) of performance? A systematic review”. *Eur Spine J* 21(4):575–598, by F. Steiger, B. Wirth, E.D. de Bruin, A.F. Mannion (2012). *European Spine Journal*. DOI: 10.1007/s00586-012-2454-1 Online First
431. Stephan, A., S. Goebel, and D. Schmidtbleicher, 2011. Effects of machine-based strength training in the therapy of chronic back pain. *German Journal of Sports Medicine*. 62, pp 69-74
432. Stevens, V. K., A. Vleeming, K. G. Bouche, N. N. Mahieu, G. G. Vanderstraeten, and L. A. Danneels, 2007. Electromyographic activity of trunk and

- hip muscles during stabilization exercises in four-point kneeling in healthy volunteers. *European Spine Journal*. 16, pp 711 – 718
433. Stevens, V. K., K. G. Bouche, N. N. Mahieu, P. L. Coorevits, G. G. Vanderstraeten, and L. A. Danneels, 2006. Trunk muscle activity in healthy subjects during bridging stabilization exercises. *BMC Musculoskeletal Disorders*. 7(75)
 434. Stevenson, J. M., C. L. Weber, J. T. Smith, G. A. Dumas, W. J. Albert, 2001. A longitudinal study of the development of low back pain in an industrial population. *Spine*. 26(12), pp 1370 – 1377
 435. Stewart, W. F., J. A. Ricci, E. Chee, D. Morganstein, and R. Lipton, 2003. Lost productive time and cost due to common pain condition in the US workforce. *JAMA*. 290(18), pp 2443 – 2454
 436. Stokes, I. A. F., S. M. Henry, and R. M. Single, 2003. Surface electrodes do not accurately record from lumbar multifidus muscles. *Clinical Biomechanics*. 18, pp 9–13
 437. Stokes, I. A. F., and J. C. Iatridis, 2004. Mechanical conditions that accelerate intervertebral disc degeneration: Overload versus immobilization. *Spine*. 29(23), pp 2724 – 2732
 438. Stone, M. H., 1990. Muscle conditioning and muscle injuries. *Medicine and Science in Sports and Exercise*. 22(4), pp 457–462
 439. Stone, W. J., and S. P. Coulter, 1994. Strength/endurance effects from three resistance training protocols with women. *Journal of Strength and Conditioning Research*. 8, pp 231–234
 440. Stothart, J. P., McGill, S. M., 2000. Stadiometry: on measurement technique to reduce variability in spine shrinkage measurement. *Clinical Biomechanics* 15(7), 546-548
 441. Suter, E., and D. Lindsay, 2001. Back muscle fatigability is associated with knee extensor inhibition in subjects with low back pain. *Spine*. 26(16), pp E361–366

442. Suuden, E., J. Erelina, H. Gapeyeva, M. Paasuke, 2008. Low back muscle fatigue during Sorenson endurance test in patients with chronic low back pain: relationship between electromyographic spectral compression and anthropometric characteristics. *Electromyography and Clinical Neurophysiology*. 48(3-4), pp 185 – 192
443. Suzuki, N., and S. Endo, 1983. A quantitative study of trunk muscle strength and fatigability in the low back pain syndrome. *Spine*. 8(1), pp 69 – 74
444. Sward, L., M. Hellstrom, B. Jacobsson, and L. Peterson, 1990. Back pain and radiologic changes in the thoraco-lumbar spine of athletes. *Spine*. 15(2), pp 124–129
445. Syczewska, M., T. Oberg, and D. Karlsson, 1999. Segmental movements of the spine during treadmill walking with normal speed. *Clinical Biomechanics*. 14, pp 384 – 388
446. Takemasa, R., H. Yamamoto, and T. Tani, 1995. Trunk muscle strength in and effect of trunk muscle exercises for patients with chronic low back pain. The differences in patients with and without organic lumbar lesions. *Spine*. 20(23), pp 2522 – 2530
447. Thépaut-Mathieu, C., J. Van Hoecke, and B. Maton, 1988. Myoelectrical and mechanical changes linked to length specificity during isometric training. *Journal of Applied Physiology*. 64(4), pp 1500 – 1505
448. Thorstensson, A., and A. Arvidson, 1982. Trunk muscle strength and low back pain. *Scandinavian Journal of Rehabilitation Medicine*. 14(2), pp 69 – 75
449. Thorstensson, A., H. Carlson, M. R. Zomlefer, and J. Nilsson, 1982. Lumbar back muscle activity in relation to trunk movements during locomotion in man. *Acta Physiologica Scandinavica*. 116, pp 13 – 20
450. Toyone, T., K. Takahashi, H. Kitahara, M. Yamagata, M. Murakami, and H. Moriya, 1994. Vertebral bone marrow changes in degenerative lumbar disc disease.

- An MRI study of 74 patients with low back pain. *The Journal of Bone and Joint Surgery*. 76(5), pp 757 – 764
451. Tsao, H., and P. W. Hodges, 2008. Persistence of improvements in postural strategies following motor control training in people with recurrent low back pain. *Journal of Electromyography & Kinesiology*. 18 (4), pp 559 – 567
452. Tucci, J. T., D. M. Carpenter, M. L. Pollock, J. E. Graves, and S. H. Leggett, 1992. Effect of reduced frequency of training and detraining on lumbar extension strength. *Spine*. 17(2), pp 1497 – 1501
453. Turk, D. C., and A. Okifuji, 2002. Psychological factors in chronic pain: evolution and revolution. *Journal of Consulting and Clinical Psychology*. 70(3), pp 678-690
454. Tyrell, A. R., Reilly, T., Troup, J. D., 1985. Circadian variation in stature and the effects of spinal loading. *Spine*. 10(2), 161-164
455. Udermann, B. E., J. E. Graves, R. G. Donelson, L. Ploutz-Snyder, J. P. Boucher, and J. H. Iriso, 1999. Pelvic restraint effect on lumbar gluteal and hamstring muscle electromyographic activation. *Archives of Physical Medicine and Rehabilitation*. 80, pp 428 – 431
456. Udermann, B. E., J. M. Mayer, J. E. Graves, and S. R. Murray, 2003. Quantitative assessment of lumbar paraspinal muscle endurance. *Journal of Athletic Training*. 38, pp 259 – 262
457. Udermann, B. E., J. M. Mayer, R. G. Donelson, J. E. Graves, and S. R. Murray, 2004. Combining lumbar extension training with McKenzie therapy: Effects on pain, disability, and psychosocial functioning in chronic low back pain patients. *Gundersen Lutheran Medical Journal*. 3(2), pp 7 – 12
458. Van Der Hoorn, W., S. M. Bruijn, O. G. Meijer, P. W. Hodges, and J. Van Dieen, 2012. Mechanical coupling between transverse plane pelvis and thorax

rotations during gait is higher in people with low back pain. *Journal of Biomechanics*. 45, pp 342 – 347

459. Van Der Hulst, M., M. M. Vollenbroek-Hutten, J. S. Rietman, and H. J. Hermens, 2010^b. Lumbar and abdominal muscle activity during walking in subjects with chronic low back pain: support of the “guarding” hypothesis? *Journal of Electromyography and Kinesiology*. 20(1), pp 31 – 38
460. Van Der Hulst, M., M. M. Vollenbroek-Hutten, J. S. Rietman, L. Schaake K. G. Groothuis-Oudshoorn, and H. J. Hermens, 2010^a. Back muscle activation patterns in chronic low back pain during walking: a “guarding” hypothesis. *Clinical Journal of Pain*. 26(1), pp 30 – 37
461. Van Der Hulst, M., M. M. Vollenbroek-Hutten, K. M. Schreurs, J. S. Rietman, and H. J. Hermens, 2010^c. Relationships between coping strategies and lumbar muscle activity in subjects with chronic low back pain. *European Journal of Pain*. 14(6), pp 640 – 647
462. Van Dieen, J. H., P. P. Kuijer, A. Burdorf, W. S. Marras, and M. A. Adams, 2012. Non-specific low back pain. *Lancet*. 379(9829), pp 1874
463. Van Dieen, J. H., and Toussaint, H. M., 1993. Spinal shrinkage as a parameter of functional load. *Spine*. 18(11), 1504-1514
464. Van Dieen, J. H., P. Van Der Burg, T. A. J. Raaijmakers, and H. M. Toussaint, 1998. Effects of repetitive lifting on kinematics: inadequate anticipatory control or adaptive changes. *Journal of Motor Behaviour*. 30(1), pp 20 – 32
465. Van Tulder, M. W., A. Malmivaara, R. Esmail, and B. W. Koes, 2000. Exercise therapy for low back pain. *The Cochrane Database of Systematic Reviews*. 2, CD00335
466. Van Tulder, M. W., B. W. Koes, and L. M. Bouter, 1995. A cost-of-illness study of back pain in The Netherlands. *Pain*. 62(2), pp 233 – 240

467. Verbunt, J. A., R. J. Smeets, and H. M. Wittink, 2010. Cause or effect? Deconditioning and chronic low back pain. *Pain*. 149, pp 428-430
468. Verbunt, J. A., K. R. Westerterp, G. J. Van Der Heijden, H. A. Seelen, J. W. Vlaeyen, and J. A. Knottnerus, 2001. Physical activity in daily life in patients with chronic low back pain. *Archives of Physical Medicine and Rehabilitation*. 82(6), pp 726–730
469. Verbunt, J. A., H. A. Seelen, J. W. Vlaeyen, G. J. van de Heijden, P. H. Huets, K. Pons, and J. A. Knottnerus, 2003. Disuse and deconditioning in chronic low back pain: concepts and hypotheses on contributing mechanisms. *European Journal of Pain*. 7(1), pp 9 – 21
470. Verna, J. L., J. M. Mayer, V. Mooney, E. A. Pierra, V. L. Robertson, J. E. Graves, 2002. Back extension endurance and strength: the effect of variable-angle roman chair exercise training. *Spine*. 27(16), pp 1772 - 1777
471. Vestergaard-Poulsen, P., C. Thomsen, T. Sinkjaer, M. Stubgaard, A. Rosenfalck, and O. Henriksen, 1992. Simultaneous electromyography and ³¹P nuclear magnetic resonance spectroscopy - with application to muscle fatigue. *Electroencephalography and Clinical Neurophysiology*. 85(6), pp 402-411
472. Vezina, M. J., and C. L. Hubley-Kozey, 2000. Muscle activation in therapeutic exercise to improve trunk stability. *Archives of Physical Medicine and Rehabilitation*. 81, pp 1370 – 1379
473. Videmann, T., M. C. Battie, K. Gill, H. Manninen, L. E. Gibbons, and L. D. Fisher, 1995. Magnetic resonance imaging findings and their relationships in the thoracic and lumbar spine: Insights into the etiopathogenesis of spinal degeneration. *Spine*. 20(8), pp 928 – 935
474. Vincent, H. K., K. R. Vincent, A. N. Seay, B. P. Conrad, R. W. Hurley, and S. Z. George, 2013. Back strength predicts walking improvement in obese, older adults with chronic low back pain. *PM & R*. 6(5), pp 418-426

475. Vincent, H. K., S. Z. George, A. N. Seay, K. R. Vincent, and R. W. Hurley, 2014. Resistance exercise, disability, and pain catastrophizing in obese adults with back pain. *Medicine and Science in Sports and Exercise*. 46(9), pp 1693-1701
476. Vindigni, D., B. F. Walker, J. R Jamison, C. Da Costa, L. Parkinson, and S Blunden, 2005. Low back pain risk factors in a large Australian aboriginal community: An opportunity for managing co-morbidities? *Chiropractic & Osteopathy*. 13(21),
477. Vindigni, D., D. Griffen, J. Perkins, C. Da Costa, and L Parkinson, 2004. Prevalence of musculoskeletal conditions, associated pain and disability and the barriers to managing these conditions in a rural, Australian aboriginal community. *Rural and Remote Health*. 4
478. Vogt, L., and W. Banzer, 1999. Measurement of lumbar spine kinematics in incline treadmill walking. *Gait & Posture*. 9(1), pp 18 – 23
479. Vogt, L., K. Pfeifer, and W. Banzer, 2003. Neuromuscular control of walking with chronic low back pain. *Manual Therapy*. 8(1). Pp 21 – 28
480. Vogt, L., K. Pfeifer, M. Portscher, and W. Banzer, 2001. Influences of nonspecific low back pain on three-dimensional lumbar spine kinematics in locomotion. *Spine*. 26(17), pp 1910 – 1919
481. Waddell, G., and A. K. Burton, 2000. Occupational health guidelines for the management of low back pain at work: evidence review. *Occupational Medicine*. 51, pp 126 – 135
482. Waddell, G., G. Feder, and M. Lewis, 1997. Systematic reviews of bed rest and advice to stay active for acute low back pain. *British Journal of General Practice*. 47, pp 647 – 652
483. Waddell, G., M. Aylward, and P. Sawney, 2002. *Back Pain, incapacity for work and social security benefits: an international literature review and analysis*. Glasgow: The Royal Society of Medicine Press Ltd

484. Wakeling, J. M., S. A. Pascual, B. M. Nigg, and V. von Tschamer, 2001. Surface EMG shows distinct populations of muscle activity when measured during sustained submaximal exercise. *European Journal of Applied Physiology*. 86, pp 40–47
485. Walker, B. F., 2000. The prevalence of low back pain: a systematic review of the literature from 1966 to 1998. *Journal of Spinal Disorders*. 13(3), pp 205 – 217
486. Walsh, A. J., and J. C. Lotz, 2004. Biological response of the intervertebral disc to dynamic loading. *Journal of Biomechanics*. 37(3), pp 329 – 337
487. Walsh, T. R., J. N. Weinstein, K. F. Spratt, T. R. Lehmann, C. Aprill, and H. Sayre, 1990. Lumbar discography in normal subjects. A controlled, prospective study. *Journal of Bone & Joint Surgery*. 72, pp 1081 – 1088
488. Walsworth, M., 2004. Lumbar paraspinal electromyographic activity during trunk exercises on two types of exercise machines. *Electromyographic and Clinical Neurophysiology*. 44, pp 201 – 207
489. Wang, D. L., S. D. Jiang, and L. Y. Dai, 2007. Biologic response of the intervertebral disc to static and dynamic compression in vitro. *Spine*. 32(23), pp 2521 – 2528
490. Watson, P. J., C. K. Booker, and C. J. Main, 1997. Evidence for the role of psychological factors in abnormal paraspinal activity in patients with chronic low back pain. *Journal of Musculoskeletal Pain*. 5(4), pp 41–56
491. Weber, B. R., D. Grob, J. Dvorak, and M. Mutener, 1997. Posterior surgical approach to the lumbar spine and its effect on the multifidus muscle. *Spine*. 22(15), pp 1765–1772
492. Weishaupt, D., M. Zanetti, J. Hodler, and N. Boos, 1998. MR imaging of the lumbar spine: prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities and osteoarthritis of the facet joints in asymptomatic volunteers. *Radiology*. 209(3), pp 661 - 669

493. Werneke, M. W., D. L. Hart, G. Cutrone, D. Oliver, T. McGill, J. Weinberg, D. Grigsby, W. Oswald, and J. Ward, 2011. Association between directional preference and centralization in patients with low back pain. *Journal of Orthopaedic and Sports Physical Therapy*. 41(1), pp 22 – 31
494. White, A. A., and S. L. Gordon, 1982. Synopsis: Workshop on idiopathic low back pain. *Spine*. 7, pp 141 – 149
495. Whiting, W. C., and R. F. Zernicke, 1998. *Biomechanics of Musculoskeletal Injury*. Human Kinetics: Champaign
496. Whitley, E., and J. Ball, 2002. Statistics review 4: Sample size calculations. *Critical Care*. 6, pp 335 – 341
497. Wilby, J., Linge, K., Reilly, T., Troup, J. D. G., 1987. Spinal shrinkage in females: circadian variation and the effects of circuit weight-training. *Ergonomics*. 30(1), 47-54
498. Wilkinson, M., 2013. Distinguishing between statistical significance and practical/clinical meaningfulness using statistical inference. *Sports Medicine*. 44(3), pp 295-301
499. Willard, F. H., 2007. The muscular, ligamentous and neural structure of the low back and its relation to back pain. In: A. Vleeming, V. Mooney, R. Stoeckart, editors. *Movement, Stability and Low Back Pain: Integration of Research and Therapy*. Churchill Livingstone: Edinburgh, pp 85–94
500. Willard, F. H., A. Vleeming, M. D. Schuenke, L. Danneels, and R. Schleip, 2012. The thoracolumbar fascia: anatomy, function and clinical considerations. *Journal of Anatomy*. 221(6), pp 507-535
501. Willemink, M. J., H. W. van Es, P. H. Helmhout, A. J. Diederik, J. Kelder, and J. P. M. van Heesewijk, 2012, The effects of dynamic isolated lumbar extensor training on lumbar multifidus functional cross-sectional area and functional status of patients with chronic nonspecific low back pain. *Spine*. 37(26)

502. Winter, D. A., 1983. Biomechanical motor patterns in normal walking. *Journal of Motor Behaviour*. 15(4), pp 302 - 330
503. Winter, D. A., C. D. MacKinnon, G. K. Ruder, and C. Wieman, 1993. An integrated EMG/biomechanical model of upper body balance and posture during human gait. *Progress in Brain Research*. 97, pp 359 – 367
504. Wittink, H., T. H. Michel, A. Wagner, A. Sukiennik, and W. Rogers, 2000. Deconditioning in patients with chronic low back pain. Fact or fiction? *Spine*. 25(17), pp 2221 – 2228
505. Wognum, S., J. M. Huyghe, and F. P. Baaijens, 2006. Influence of osmotic pressure changes on the opening of existing cracks in 2 intervertebral disc models. *Spine*. 31 (19), pp 1783 – 1788
506. Woolf, C. J., and M. W. Salter, 2000. Neuronal plasticity: Increasing the gain in pain. *Science*. 288, pp 1765 – 1768
507. Woolf, C. J., G. J. Bennett, M. Doherty, R. Dubner, B. Kidd, M. Koltzenburg, R. Lipton, J. D. Loeser, R. Payne, and E. Torebjork, 1998. Towards a mechanism-based classification of pain? *Pain*. 77(3), pp 227 – 229
508. World Health Organisation (WHO), 1998. *The World Health Report 1998: Life in the 21st century: A vision for all*. Geneva: Office of Publications, World Health Organisation
509. Yoshihara, K., Y. Shirai, Y. Nakayama, and S. Uesaka, 2001. Histochemical changes in the multifidus muscle in patients with lumbar intervertebral disc herniation. *Spine*. 26(6), pp 622–626
510. Zander, G., 1872. OM Medico-Mekaniska Instituteti Stockholm. *Journal of Nordic Medicine*. Band IV
511. Zhao, F., P. Pollintine, B. D. Hole, P. Dolan, and M. A. Adams, 2005. Discogenic origins of spinal instability. *Spine*. 30(23), pp 2621 – 2630

512. Zhao, W. P., Y. Kawaguchi, H. Matsui, M. Kanamori, and T. Kimura, 2000.
Histochemistry and morphology of the multifidus muscle in lumbar disc herniation: a comparative study between diseased and normal sides. *Spine*. 25(17), pp 2191 – 2199
513. Zhu, X. Z., M. Parnianpour, M. Nordin, and N. Kahanovitz, 1989.
Histochemistry and morphology of erector spinae muscle in lumbar disc herniation. *Spine*. 14(4), pp 391–397

7.0 APPENDICES

7.1 Published Research

This appendix presents published work by the research by the author. A list of both peer reviewed journal articles, book chapters and also conference presentations is provided.

7.1.1 Peer Reviewed Journal Articles

- Fisher, J., and **J. Steele**, 2014. Questioning the resistance/aerobic training dichotomy: A commentary on physiological adaptations determined by effort rather than exercise modality. *Journal of Human Kinetics*. 44, pp 137-142
- **Steele, J.**, S. Bruce-Low, and D. Smith, 2014. A review of the clinical value of isolated lumbar extension resistance training for chronic low back pain. *PM&R*. In press
- Giessing, J., J. Fisher, **J. Steele**, F. Rothe, K. Raubold, and B. Eichmann, 2014. The effects of low volume resistance training with and without advanced techniques in trained participants. *Journal of Sports Medicine and Physical Fitness*. In press
- **Steele, J.**, S. Bruce-Low, D. Smith, D. Jessop, and N. Osborne, 2014. Lumbar kinematic variability during gait in chronic low back pain and associations with pain, disability and isolated lumbar extension strength. *Clinical Biomechanics*. In press
- Fisher, J., **J. Steele**, P. McKinnon, and S. McKinnon, 2014. Strength gains as a result of brief, infrequent resistance exercise in older adults. *Journal of Sports Medicine*. Pp 1-7
- **Steele, J.**, S. Bruce-Low, D. Smith, N. Osborne, and A. Thorkeldsen, 2014. Can specific loading through exercise impart healing or regeneration of the intervertebral disc? *The Spine Journal*. In Press
- Fisher, J., L. Carlson, **J. Steele**, and D. Smith, 2014. Effects of pre-exhaustion, exercise order and rest-intervals in a full body resistance training intervention in

trained participants. *Applied Physiology, Nutrition and Metabolism*. Epub ahead of print

- Fisher, J., **J. Steele**, M. Brzycki, and B. DeSimone, 2014. Primum non nocere: A commentary on avoidable injuries and safe resistance training techniques. *Journal of Trainology*. 3(1), pp 31-34
- **Steele, J.**, and J. Fisher, 2014. Scientific rigour: a heavy or light load to carry? *Sports Medicine*. 44(1), pp 141 – 142
- **Steele, J.**, S. Bruce-Low, and D. Smith, 2014. A Reappraisal of the Deconditioning Hypothesis in Low Back Pain: Evidence from a Triumvirate of Research Methods on Specific Lumbar Extensor Deconditioning. *Current Medical Research and Opinion*. 30(5), pp 865-911
- Fisher, J., **J. Steele**, and D. Smith, 2013. Evidence Based Resistance Training Recommendations for Muscular Hypertrophy. *Medicina Sportiva*. 17(4), pp 217 – 235
- **Steele, J.**, S. Bruce-Low, and D. Smith, 2013. A review of the specificity of exercises designed for conditioning the lumbar extensors. *British Journal of Sports Medicine*. Online First
- **Steele, J.**, S. Bruce-Low, and D. Smith, 2013. Controlling Resistance Training Variables in Interventions for Chronic Nonspecific Low Back Pain: Letter to the Editor regarding “The Effects of Dynamic Isolated Lumbar Extensor Training on Lumbar Multifidus Functional Cross-Sectional Area and Functional Status of Patients with Chronic Nonspecific Low Back Pain.” *Spine*. 38(18), pp 1609 – 1610
- **Steele, J.**, S. Bruce-Low, D. Smith, D. Jessop, and N. Osborne, 2013. A Randomised Controlled Trial of Limited Range of Motion Lumbar Extension Exercise in Chronic Low Back Pain. *Spine*. In Press
- **Steele, J.**, 2013. Intensity; in-ten-si-ty; noun. 1. Often used ambiguously within resistance training. 2. Is it time to drop the term altogether? *British Journal of Sports Medicine*. Online First

- **Steele, J.**, J. Fisher, D. McGuff, S. Bruce-Low, and D. Smith, 2012. Resistance training to momentary muscular failure improves cardiovascular fitness in humans: A review of acute physiological responses and chronic physiological adaptations. *Journal of Exercise Physiology*. 15(3), pp 53 – 80
- **Steele, J.**, and S. Bruce-Low, 2012. Steiger et al. 2011: relationships and specificity in CLBP rehabilitation through exercise. *European Spine Journal*. 21(9), pp 1887
- Fisher, J., and **J. Steele**, 2012. Is truth in authority or authority in truth? Limitations to the publication of scientific research. *Journal of Exercise Physiology*. 15(1), pp 57 – 64
- Fisher, J., **J. Steele**, S. Bruce-Low, and D. Smith, 2011. Evidence Based Resistance Training Recommendations. *Medicina Sportiva*. 15(3), 147 – 162

7.1.2 Conference Presentations

- **Steele, J.**, 2014. A synthesis of modern exercise physiology and evolutionary theory. Presented at the *Ancestral Health Symposium*, University of California, Berkeley. Available at: <https://www.youtube.com/watch?v=8YFdL7D65Ng>
- **Steele, J.**, S. Bruce-Low, D. Smith, D. Jessop, and N. Osborne, 2013. Isolated lumbar extension resistance reduces lumbar kinematic variability during gait in chronic low back pain participants. Presented at *World LBP Congress*, Dubai Intercontinental and Crowne Plaza Hotels
- **Steele, J.**, 2013. An Ancient Perspective on Deconditioning in Low Back Pain. Presented at *Ancestral Health Symposium*, Atlanta Sheraton Hotel
- **Steele, J.**, S. Bruce-Low, D. Smith, and D. Jessop, 2012. Limited Range of Motion Lumbar Extension Resistance Exercise in Chronic Low Back Pain Participants. Presented at *Physiology*, Edinburgh International Conference Centre
- **Steele, J.**, and S. Bruce-Low, 2010. Effect of training with and without pelvic restraints, on development of lumbar extension strength and lumbar muscle activity.

Presented at *British Association of Sport and Exercise Science Student Conference*,
University of Aberystwyth

- **Steele, J.**, and S. Bruce-Low, 2009. Effect of training with and without pelvic restraints, on development of lumbar extension strength and lumbar muscle activity – A Research Proposal. Presented at *British Association of Sport and Exercise Science Student Conference*, University of Hull

7.2 Top Lesson(s) Learnt from Undertaking the Ph.D

Prior to my *viva* this section constituted a lengthy personal reflection of some of the key lessons I learnt during the process of undertaking the Ph.D. It contained lengthy diatribes about some of the more frustrating elements of the process along with lessons learnt about time management, prioritisation, peer review, 'playing the game', writing and presenting research, logistics of applied research, adaptability etc. Writing it was very difficult. It felt strange to put down many of these thoughts in a personal manner into what, for the past few years, has been a very formal document for me. I even spent weeks in trying to write it searching for examples from other Ph.D students to see exactly what was supposed to go into it. Since completing my *viva* and it being suggested that I reduce this section to a shorter 'lessons learnt' I returned to these examples and found to my surprise the congruence between what I had learnt and what many others had. There was however one lesson I felt I had learnt that was ultimately missing from the examples I could find. So in order to keep this a short 'lessons learnt' I thought I would dedicate it to the biggest lesson I felt I learnt from undertaking the Ph.D

It's not the end.....merely the beginning

I was discussing the writing of this section with a colleague before I began it and explained the difficulty I was having. I felt I could identify my reflections internally and potentially verbalise them but would struggle in written form. He offered some advice and noted how he personally thought I had matured intellectually from when I completed my undergraduate and began my Ph.D. What I found curious was how obvious to me this was *now*. But if I really think about it when I look back, in the beginning I felt the same way I do now, or at least very similar. Back then I only had the past to compare to and logically I couldn't know what I didn't know. I felt like I knew what I was doing. Today I still feel like that. Back then it was most definitely misguided and even today probably still so. But when I compare back between today and then I can see the glaring difference. In a way however I do feel different in one respect. Though I feel confident in my knowledge and the way I have gone about

obtaining it, knowing its strengths and its weaknesses, in reflection I am now more acutely aware and have appreciation of the fact that, even though my knowledge grows, there are still, and always will be, things I do not know. For some that's quite scary. For me and many others it's quite exciting. Even as my knowledge grows I will never run out of interesting problems to look at.

Trying to put together a personal reflection helped me to understand more what a Ph.D really is – what the biggest lesson to be learnt from the process is (at least for me). For much of my time spent engaged in the process I had been attempting to produce a piece of work that most people would have spent a lifetime putting together, through meticulous examination of the literature from all perspectives, and through conducting a considerable amount of the empirical work themselves. In the beginning I had the belief that my Ph.D was to be my single *magnus opus*. Instead I have come to realise that, in a way it would be, but only for a short time. Relative to all work I have done up until this point it is indeed my greatest achievement and there is nothing wrong with being proud of that. However, with hindsight we are always able to see that, in comparison to what we were, we continue to grow and develop. My Ph.D was a concerted effort in a very specific area. My '*real*' *magnus opus* was to always and inevitably come later, again and again. My Ph.D, though a great achievement, has been a stepping stone upon which I have honed my research skills in a specific arena in order to go out into the world and begin to use them to solve its myriad problems. Sure, I learnt all the *cliché* things that most people put into personal reflections of this process (time management, prioritisation, peer review, 'playing the game', writing and presenting research, logistics of applied research, adaptability etc.). I made mistakes; I failed along the journey and learnt from this. But the biggest lesson I realised I had learnt as I completed the journey was to maintain an appropriate degree of humility coupled with the determination to progress further. In essence, that the Ph.D isn't the end....it's only the beginning.

Centre for
Health
Exercise and
Sport
Science



Confirmation of HESS Ethical Clearance

17/11/2010

To whom it may concern,

This letter is to inform that the research project entitled "Effect of limited range of motion lumbar extension training on lumbar strength, gait variability and pain in symptomatic chronic lower back pain participants" that is being conducted by student researcher Mr James Steele and supervised by Dr Stewart Bruce-Low has been approved and cleared through the Health, Exercise and Sport Science ethical guidelines for approval.

Yours Faithfully,

A handwritten signature in blue ink that reads "Patricia D. Park".

Professor Patricia Park



National Research Ethics Service
NRES Committee South Central - Southampton B

Level 3 Block B
Whitefriars
Lewins Mead
Bristol
BS1 2NT

Telephone: 0117 3421384
Facsimile: 0117 3420445

Dr Stewart Bruce-Low
Senior Lecturer & Laboratory Director
Southampton Solent University
Sport Science Department
East Park Terrace
Southampton SO14 0YN

03 May 2011

Dear Dr Bruce-Low

Study title: Effect of limited range of motion lumbar extension training on lumbar strength, gait variability and pain in symptomatic chronic lower back pain participants.

REC reference: 11/H0504/9

Thank you for your letter responding to the Committee's request for further information on the above research and submitting revised documentation.

The further information was considered in correspondence by a sub-committee of the REC at a meeting held on 27 April 2011. A list of the sub-committee members is attached.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a **favourable** ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation [as revised], subject to the conditions specified below.

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission ("R&D approval") should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at <http://www.rdforum.nhs.uk>.

Where a NHS organisation's role in the study is limited to identifying and referring potential participants to research sites ("participant identification centre"), guidance should be sought from the R&D office on the information it requires to give permission for this activity.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of approvals from host organisations

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

<i>Document</i>	<i>Version</i>	<i>Date</i>
Covering Letter		07 January 2011
Summary/Synopsis	Protocol Flow Diagram	07 January 2011
Letter from Sponsor		17 November 2010
Investigator CV	Dr Stewart Bruce-Low	07 January 2011
Questionnaire: Revised Oswestry Questionnaire	2	07 January 2011
REC application		10 January 2011
Participant Consent Form	1	07 January 2011
Unfavourable Opinion Letter from Oxfordshire REC A		14 January 2011
Confirmation of HESS Ethical Clearance		17 November 2010
Comments and Changes made	1	17 March 2011
Questionnaire: A2 Physical Activity Readiness Questionnaire	2	07 January 2011
Advertisement	Poster version 2	07 January 2011
Response to points raised by Oxfordshire REC A		
CV - Sally Matthews		
CV - Mr James Steele		
GP/Health Professional Referral Form	2	07 January 2011
CV - Gary Bissell		
GP/Health Professional Notification Letter	3	17 March 2011
GP/health Professional Referral Form	3	17 March 2011
CV Neil Osborne (Chiropractor)	1	17 March 2011
Letter of invitation to participant	Email version 2	07 January 2011
GP/Consultant Information Sheets	2	07 January 2011
Participant Recruitment Flow Diagram	2	07 January 2011
Visual Analogue Pain Scale	1	17 March 2011
Evidence of insurance or indemnity		04 August 2010
Letter from Statistician	Scott Burnet	07 January 2011
Protocol	2	07 January 2011
Response to Request for Further Information		
Participant Information Sheet	3	17 March 2011

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Now that you have completed the application process please visit the National Research Ethics Service website > After Review

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

The attached document "*After ethical review – guidance for researchers*" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

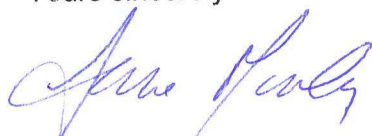
We would also like to inform you that we consult regularly with stakeholders to improve our service. If you would like to join our Reference Group please email referencegroup@nres.npsa.nhs.uk.

11/H0504/9

Please quote this number on all correspondence

With the Committee's best wishes for the success of this project

Yours sincerely



Dr Helen McCarthy
Chair

Email: scsha.swrecb@nhs.net

Enclosures: *List of names and professions of members **who were present at the meeting***

"After ethical review – guidance for researchers" [SL-AR2]

Copy to: *Dr Stewart Bruce-Low*

NRES Committee South Central - Southampton B

Attendance at Sub-Committee of the REC meeting on 27 April 2011

Professor Ron King
Mrs Janet Brember

PARTICIPANT INFORMATION SHEET

This research study is being completed as part of a PhD by Mr James Steele under the supervision of Dr Stewart Bruce-Low

Study Title: Effect of limited range of motion lumbar extension training on lumbar strength, gait variability and pain in symptomatic chronic lower back pain participants.

Invitation to participate

We would like to invite you to take part in our research study. Before you decide we would like you to understand why the research is being done and what involvement you would have, so you are asked to read this form carefully. One of our team will be able to go through the information sheet with you and answer any questions you have. We suggest you take time over the following week to read and digest the material. We also advise that you talk to others about the study if you wish. After receipt of this information sheet and having read and understood it, we ask that you respond within a period of one week as to your intent to participate. If we do not receive a reply within 7 days we will consider that you no longer wish to become a participant in the research. If you consent to take part, as a participant, in the study being undertaken by James Steele, then you should sign the consent form. If you have any query, or are unsure or uncertain about anything, then you should not sign until your query has been resolved and you are completely happy to volunteer.

What is the purpose of the study?

The study wishes to investigate whether it is possible to produce improvement in strength of the muscles in your lower back by undertaking exercise that limits your movements and in turn wants to observe if strength increases can still occur throughout your full range of movement. In addition the study wishes to investigate whether this training has any effect upon the way in which you walk (also known as 'gait').

Why have I been invited to participate?

You have been invited to take part in the study as you have non-specific chronic low back pain and you do not have any of the exclusion criteria (listed in the section below).

Exclusion Criteria

You must have no medical condition for which resistance training would be contraindicated. Some examples of these medical conditions may include: acute (not re-occurring) low back injury occurring within the last 12 weeks, pregnancy, sciatic nerve root compression (sciatica), severe disc herniation (slipped disc), previous vertebral fractures (fractures in the bones of your spine) or major structural abnormalities. If you have any of these symptoms you will not be able to participate in the project because, in the opinion of the researchers, it may involve an unacceptable risk to yourself. If you are unsure of whether you fall into any of these categories it is not a problem as we will require you to have a referral form completed by a GP/Health Professional who can verify this and that you are free from any other condition which in their opinion would prevent you from participating in the study. If the referring GP/Health Professional deems you unfit for the study then you will be excluded from participation. Please note that the health professional may require a charge for referring you.

Do I have to take part?

It is up to you to decide to join the study. We will describe the study and go through this information sheet. If you agree to take part, we will then ask you to sign a consent form. You are free to withdraw at any time, without giving reason. This would not affect the standard of care you receive.

What are the possible benefits of taking part?

The potential benefits of taking part in the research are to increase your range of movement and the strength of the muscles in your low back and also improve the way in which you walk (gait). We will also try to reduce the amount of pain you are currently experiencing.

Centre for
Health
Exercise and
Sport
Science



What are the possible disadvantages and risk of taking part?

As with any form of exercise there is potential for injury to occur through the use of the machine or it may worsen your condition. This likelihood is minimised through you being referred by either a GP, chiropractor or physiotherapist who will state you are ok to exercise; checking your current health status and using an effective warm up and cool down during each exercise session by the researchers.

What will I be asked to do during the study?

Upon recruitment to the study you will be randomly assigned to 1 of a possible 3 groups, 1) a group that will perform resistance exercise for the low back muscles throughout a limited range of movement (see picture below), 2) a group that will perform resistance exercise for the lumbar extensors throughout their full range of movement (see picture below), or 3) a control group that will not perform any training. Throughout the study it is recommended that you continue with any current treatments you may be utilizing, though it is recommended that you avoid any additional resistance exercises that are designed to target the low back muscles specifically and in isolation.

Prior to starting the study you will be asked to supply all your previous information regarding your condition (this may include; case notes, diagnoses and scans if appropriate). This information may be passed (although your name will be removed from all paper work so you can not be identified) to a chiropractor for further examination if the research team deems it appropriate. If you are appropriate to join the study and do not meet the exclusion criteria, you will be asked to go to the Centre for Health, Exercise and Sport Science at Southampton Solent University where all testing and training will be conducted. Firstly you will be asked to complete a few questionnaires in order to assess your pain, disability and general health. You will then undertake 2 tests on separate days 72 hours apart using the MedX Lumbar Extension Machine (see picture below). This machine isolates the muscles of the lower back (see picture below) in order to test your strength and range of movement of your low back. Upon a third visit you will have your walking style tested using a special camera system. This will involve the use of adhesive markers placed upon your lower back (see picture below). After these first two testing sessions have been completed you will begin your training (the type of training will depend on which group you have been allocated to). The training will last for 12 weeks during which the groups performing exercise will attend the Centre for Health, Exercise and Sport Science once a week in order to perform a single set of resistance exercise which will exercise their low back muscles to fatigue (up until the point you cannot push against the machine in a controlled manner anymore).

Upon completion of the training you will revisit the Centre for Health, Exercise and Sport Science in order to perform all the tests you did on the first week of attending the Centre. The idea is to measure any changes due to the training you have just undertaken. The testing/training sessions will take no more than 20 minutes (training sessions in particular will involve 70-105 seconds of actual exercise), will involve trying as hard as you can which may cause some discomfort in the lower back. Also, there may be some slight soreness in the low back muscles some 48-72 hours after testing or training. These effects are perfectly normal, are not harmful and usually resolve themselves after 72 hours. Participants in the control groups may also experience this soreness after testing.

What happens when the research study stops?

When you have completed the study you will be given the option to continue with the programme of exercise if you wish, and if the researcher testing you feels it would be beneficial.

What if I wish to make a complaint?

Any complaint about the way you have been dealt with during the study or any possible harm you might suffer will be addressed. You may register any complaint you might have about this experiment to the Faculty Research Advisor, Sport, Tourism and Leisure School at Southampton Solent University. You will be offered the opportunity of providing feedback on the experiment using standard report forms.

Will my taking part in the study be kept confidential?

Yes. We will follow ethical and legal practice and all information about you will be handled in confidence.

What if relevant new information becomes available?

Sometimes we get new information about the exercise being studied. If this happens, your researcher will tell you and discuss whether you should continue in the study. If you decide not to carry on, your researcher will make arrangements for you to discontinue your sessions. If you decide to continue in the study s/he may ask you to sign an agreement outlining the discussion.

What will happen if I don't want to carry on with the study?

You can withdraw from the study at any time with out any repercussion to yourself. If you are happy to do so, your data that has been collected will still be used within the research study (although your identity will remain confidential).

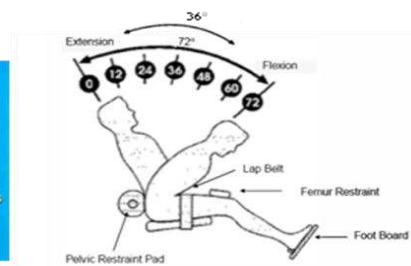
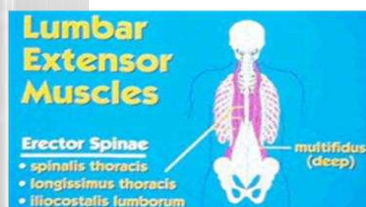
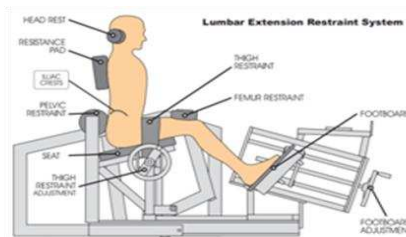
What if there is a problem?

If you have a concern about any aspect of this study, you should ask to speak to the student researcher who will do their best to answer your questions (02380319606 or research97@solent.ac.uk) or their supervisor Dr Stewart Bruce-Low (02380319272 stewart.bruce-low@solent.ac.uk). If you remain unhappy and wish to complain formally, you can do this via the Faculty Research Advisor, Sport, Leisure and Tourism School at Southampton Solent University (02380319850).

Southampton Solent University holds public liability insurance and professional indemnity to cover negligent activity resulting in the event of a trial related injury. All participants will be referred from chiropractors, GPs or physiotherapists in the local area, therefore, prior to commencement of the study you will have been cleared to exercise from the relevant health care practitioner.



Above Picture: Marker placements for gait analysis



Top Left Picture: MedX Lumbar Extension Machine

Top Right Picture: MedX Lumbar Extension Machine Schematic

Bottom Left Picture: Muscles that extend the lower back

Bottom Right Picture: Example of full range of movement and limited range of movement

Centre for
Health
Exercise and
Sport
Science



CONSENT FORM

Title of Study: Effect of limited range of motion lumbar extension training on lumbar strength, gait variability and pain in symptomatic chronic lower back pain participants

Name of Researcher: Mr James Steele

Name of Participant: _____

Please Initial Box

1. I confirm that I have read and understand the Participant Information Sheet (Version 1, dated 3rd December 2010) for the above study and have had the opportunity to ask questions.
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my treatment or legal rights being affected.
3. I agree to the storage of my personal data as it pertains to the above study and am aware that my identity will remain confidential and stored subject to the conditions of the Data Protection Act 1998.
4. I have informed the researchers of my participation in any other research study.
5. I agree to take part in the above study.

☐☐☐☐☐

Name of Participant

Date

Signature

Name of Researcher

Date

Signature

PARTICIPANT INFORMATION SHEET

This research study is being completed as part of a PhD by Mr James Steele under the supervision of Dr Stewart Bruce-Low

Study Title: The Effects of Isolated Lumbar Extension Resistance Training Upon Seated Stadiometry in Participants with Chronic Low Back Pain.

Invitation to participate

We would like to invite you to take part in our research study. Before you decide we would like you to understand why the research is being done and what involvement you would have, so you are asked to read this form carefully. One of our team will be able to go through the information sheet with you and answer any questions you have. We suggest you take time over the following week to read and digest the material. We also advise that you talk to others about the study if you wish. After receipt of this information sheet and having read and understood it, we ask that you respond within a period of one week as to your intent to participate. If we do not receive a reply within 7 days we will consider that you no longer wish to become a participant in the research. If you consent to take part, as a participant, in the study being undertaken by James Steele, then you should sign the consent form. If you have any query, or are unsure or uncertain about anything, then you should not sign until your query has been resolved and you are completely happy to volunteer.

What is the purpose of the study?

The study wishes to investigate whether it is possible to produce improvement in strength of the muscles in your lower back by undertaking exercise using a specialised machine (see picture below) and in turn wants to observe if strength increases due to this training has any effect upon lumbar intervertebral disc hydration as measured by seated height (seated stadiometry).

Why have I been invited to participate?

You have been invited to take part in the study as you have non-specific chronic low back pain and you do not have any of the exclusion criteria (listed in the section below).

Exclusion Criteria

You must have no medical condition for which resistance training would be contraindicated. Some examples of these medical conditions may include: acute (not re-occurring) low back injury occurring within the last 12 weeks, pregnancy, evidence of sciatic nerve root compression (sciatica), leg pain radiating to below the knee, paraesthesia (tingling or numbness), no current tension sign, no lower limb motor deficit, current disc herniation (slipped disc), previous vertebral fractures (fractures in the bones of your spine) or major structural abnormalities. If you have any of these symptoms you will not be able to participate in the project because, in the opinion of the researchers, it may involve an unacceptable risk to yourself. If you are unsure of whether you fall into any of these categories it is not a problem as we will require you to have a referral form completed by a Chiropractor who can verify this and that you are free from any other condition which in their opinion would prevent you from participating in the study. If the referring Chiropractor deems you unfit for the study then you will be excluded from participation. Please note that we can arrange for a Chiropractor working with the research team to perform this free of charge, however, if this is done by an external Chiropractor they may require a charge for referring you.

Do I have to take part?

It is up to you to decide to join the study. We will describe the study and go through this information sheet. If you agree to take part, we will then ask you to sign a consent form. You are free to withdraw at any time, without giving reason. This would not affect the standard of care you receive.

What are the possible benefits of taking part?

The potential benefits of taking part in the research are to increase your range of movement and the strength of the muscles in your low back and also improve the condition of your lumbar intervertebral discs. We will also try to reduce the amount of pain and disability you are currently experiencing.

What are the possible disadvantages and risk of taking part?

As with any form of exercise there is potential for injury to occur through the use of the machine or it may worsen your condition. This likelihood is minimised through you being screened by a Chiropractor who will state you are ok to exercise, and also by checking your current health status and using an effective warm up and cool down during each testing and exercise session by the researchers.

What will I be asked to do during the study?

Upon recruitment to the study you will initially be tested for a range of variables detailed below. Then you will begin a control period where you will continue with normal activities for the initial 12 weeks of the study. After these 12 weeks you will be retested and then randomly assigned to 1 of a possible 2 groups, 1) a group that will perform resistance exercise for the low back muscles using a heavy load, or 2) a group that will perform resistance exercise for the lumbar extensors using a light load. Throughout the study it is recommended that you continue with any current treatments you may be utilizing, though it is recommended that you avoid any additional resistance exercises that are designed to target the low back muscles specifically and in isolation.

Prior to starting the study and throughout if applicable, you will be asked to supply all your previous information regarding your condition (this may include; case notes, diagnoses and scans if appropriate). This information may be passed (although your name will be removed from all paper work so you can not be identified) to a chiropractor for further examination if the research team deems it appropriate. If you are appropriate to join the study and do not meet the exclusion criteria, you will be asked to go to the Centre for Health, Exercise and Sport Science at Southampton Solent University where all testing and training will be conducted. You are encouraged to avoid heavy lifting for 48 hours prior to attending testing sessions. Firstly you will be asked to complete a few questionnaires in order to assess your pain, disability and general health. You will then undertake 2 tests on separate days at least 72 hours apart using the MedX Lumbar Extension Machine (see picture below). This machine isolates the muscles of the lower back (see picture below) in order to test your strength and range of movement of your low back. During the first visit you will also have your seated height measured. This will involve the use of a specialized equipment setup (see picture below). During these sessions you will also have your seated height measured. You will be required to come in on three consecutive days (including the first visit) during the initial testing at the same time of day in order to take multiple seated height measures. After initial testing sessions (both at the beginning and end of the control period) have been completed you will begin your training (the type of training will depend on which group you have been allocated to). The training will last for 12 weeks during which the groups performing exercise will attend the Centre for Health, Exercise and Sport Science once a week in order to perform a single set of resistance exercise which will exercise their low back muscles to fatigue (up until the point you can no longer move the load applied by the machine in a controlled manner).

Upon completion of the training you will revisit the Centre for Health, Exercise and Sport Science in order to perform all the tests you did on the first week of attending the Centre. The idea is to measure any changes due to the training you have just undertaken. The testing/training sessions will take no more than 20 minutes (training sessions in particular will involve 70-105 seconds; heavy load group, or 105-140 seconds; light load group, of actual exercise), and will involve a maximal physical effort which may cause some discomfort in the lower back. Also, there may be some slight soreness in the low back muscles some 48-72 hours after testing or training. These effects are perfectly normal effects of the exercise, are not harmful and usually resolve themselves after 72 hours. Participants in the control groups may also experience this soreness after testing.

What happens when the research study stops?

When you have completed the study you will be given the option to continue with the programme of exercise if you wish, and if the researcher testing you feels it would be beneficial. However, continuation after the research has ended will involve a charge which the researcher can inform you about at the time.

What if I wish to make a complaint?

Any complaint about the way you have been dealt with during the study or any possible harm you might suffer will be addressed. You may register any complaint you might have about this experiment to the Faculty Research Advisor, Sport, Tourism and Leisure School at Southampton Solent University. You will be offered the opportunity of providing feedback on the experiment using standard report forms.

Will my taking part in the study be kept confidential?

Yes. We will follow ethical and legal practice and all information about you will be handled in confidence

What if relevant new information becomes available?

Sometimes we get new information about the exercise being studied. If this happens, your researcher will tell you and discuss whether you should continue in the study. If you decide not to carry on, your researcher will make arrangements for you to discontinue your sessions. If you decide to continue in the study s/he may ask you to sign an agreement outlining the discussion.

What will happen if I don't want to carry on with the study?

You can withdraw from the study at any time with out any repercussion to yourself. If you are happy to do so, your data that has been collected will still be used within the research study (although your identity will remain confidential).

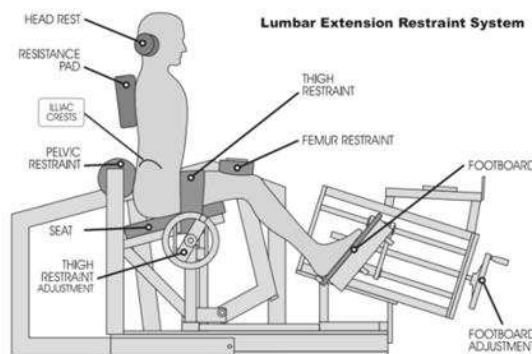
What if there is a problem?

If you have a concern about any aspect of this study, you should ask to speak to the student researcher who will do their best to answer your questions (02380319606 or james.steele@solent.ac.uk) or their supervisor Dr Stewart Bruce-Low (02380319272 stewart.bruce-low@solent.ac.uk). If you remain unhappy and wish to complain formally, you can do this via the Faculty Research Advisor, Sport, Leisure and Tourism School at Southampton Solent University (02380319850).

Southampton Solent University holds public liability insurance and professional indemnity to cover negligent activity resulting in the event of a trial related injury. All participants will be referred from chiropractors, GPs or physiotherapists in the local area, therefore, prior to commencement of the study you will have been cleared to exercise from the relevant health care practitioner.



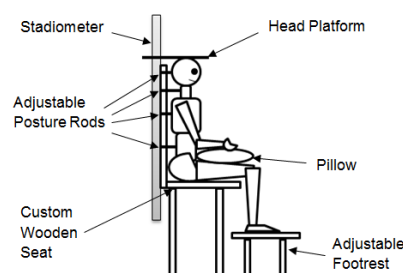
Left Picture: MedX Lumbar Extension Machine



Middle Picture: MedX Lumbar Extension Machine Schematic



Right Picture: Muscles that extend the lower back



Middle Picture: Set-up for measurement of seated height

CONSENT FORM

Title of Study: The Effects of Isolated Lumbar Extension Resistance Training Upon Seated Stadiometry in Participants with Chronic Low Back Pain.

Name of Researcher: Mr James Steele

Name of Participant: _____

Please Initial Box

1. I confirm that I have read and understand the Participant Information Sheet for the above study and have had the opportunity to ask questions.
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my treatment or legal rights being affected.
3. I agree to the storage of my personal data as it pertains to the above study and am aware that my identity will remain confidential and stored subject to the conditions of the Data Protection Act 1998.
4. I have informed the researchers of my participation in any other research study.
5. I agree to take part in the above study.

☐☐☐☐☐

Name of Participant

Date

Signature

Name of Researcher

Date

Signature

GP/Health Professional Referral Form

Dear GP/Health Professional,

This form will verify that your patient has agreed to undertake research within the Health, Exercise and Sport Science Programme at Southampton Solent University.

The test procedures are outlined on the attached Participant Information Sheet:

Please can you confirm that _____ is suitable to undertake this form of exercise test?

GP/Health Professional Name:

Physiotherapist Signature:

Any additional comments by the Physiotherapist:

Thank you for your time in this matter.

Kind regards,

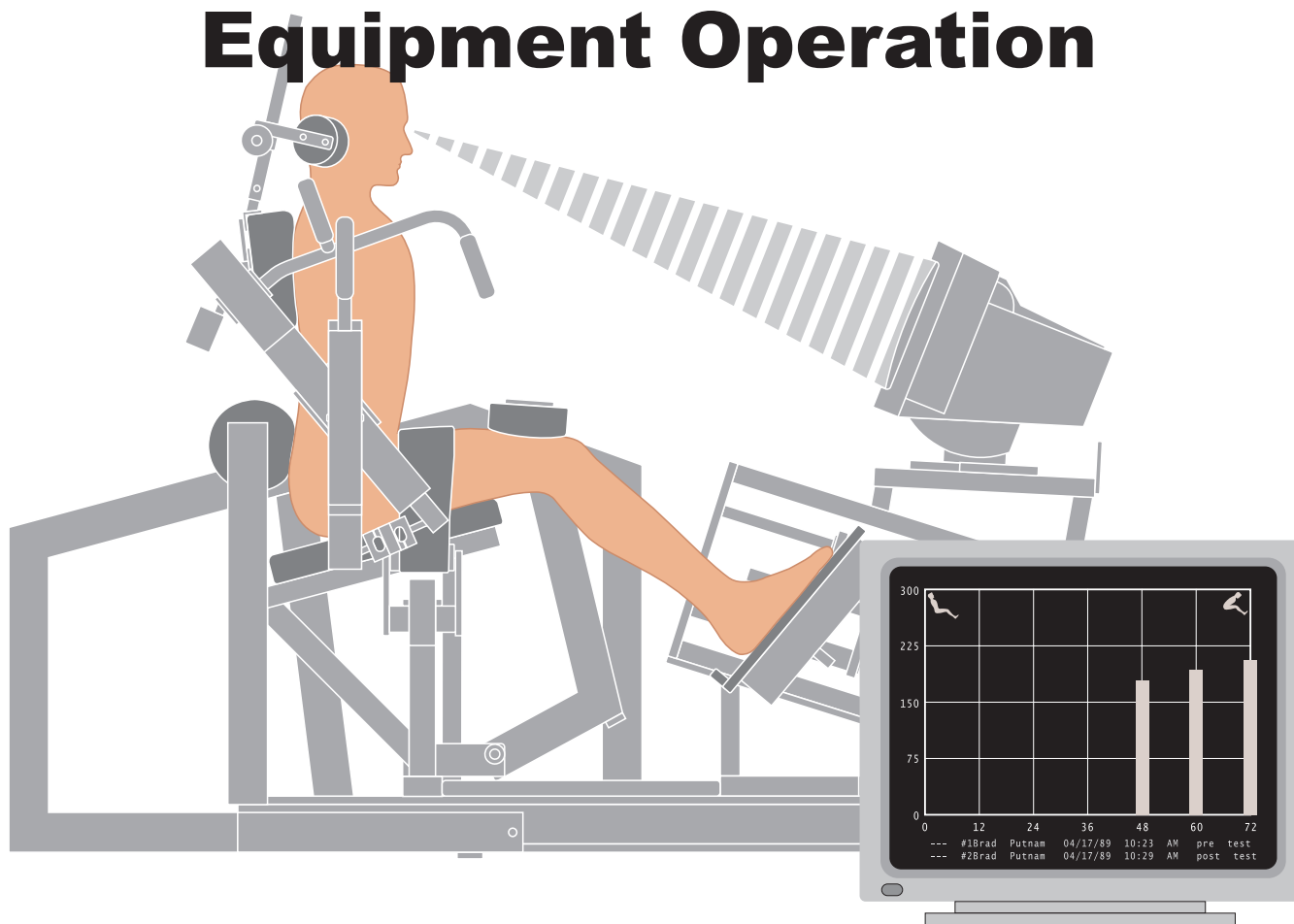
Researcher: (Print) James Steele

Signature.....

Position: PhD Research Student

A copy of this letter (once returned by the GP/Health Professional) must be kept in the faculty office.

Equipment Operation



MedX medical machines provide two phases of operation:

1. Static testing (torque readings of isometric contractions at selected angular positions).
2. Dynamic exercise (lifting and lowering a selected level of weight stack resistance).

Inherent in each of these tests is a range-of-motion assessment (ROM), which can be considered a third function of each machine.

The computer system monitors and records each of these operations, but does not control the patient in any way.

The clinician must set the patient into the machine, instruct them what to do, and use the machine's software to execute each function. The clinician can also use the software to crunch data and issue reports.

Thorough exploration of this manual is necessary to gain full command of this equipment's capabilities. This section is devoted to the bare-bone basics of mechanical operation of the equipment.

On the Lumbar Extension Machine, a computer monitor stationed atop the footboard provides visual feedback to the patient.

Checklist Exercises

The checklists that follow — 104 points on the Lumbar and 100 points on the Cervical — were adapted directly from the hands-on spinal certification test at the University of Florida.

As an exercise to assess skill with operating the machinery, it involves an isometric test followed by a dynamic exercise set, immediately followed by another isometric test. This is also known as the FRT (fatigue response test (see pages 3-9, 10). Performing this exercise successfully indicates a solid command of the technology.

104-POINT CHECKLIST

Lumbar Machine Patient Test, Exercise Sessions

PT PREPARATION

1. Greet pt., explain purpose of visit
2. Have pt. perform static stretches
3. Have pt. void, if necessary
4. Have pt. remove belt & empty pockets

CALIBRATION

5. Move to calibrate, calibration update
6. CounterWeight (CW) unlocked
7. Angle selector locked at 18°
8. Loosen chain, remove pins from weightstack
9. Pot/SG count #'s within range
[if not within range, contact
MedX Tech Support 800-528-3159]

TEST SETUP

10. Select file for test (retrieve or new)
11. Explain: -test sequence (pre/dyn/post)
 12. need for stabilization (isolation)
 13. should feel pressure from restraints
 14. should not feel pain/numbness
 15. should not feel dizzy, short of breath, etc.



The lap belt, left, and thigh restraint, right.

Lumbar Machine Patient Test, Exercise Sessions (cont.)



16. Align iliac crests wnh centerline of pelvic restraints
17. Secure thigh restraint
18. Select femur restraint ht. (legs parallel to seat; 15°)
19. Position feet on footboard (toes turned inward)
20. Secure femur restraint (heels $\leq 1/2$ inch)
21. Check for pelvic rotation (pt. reach for toes)
22. Adjust head pad (base of occipital bone)
23. Find max angle extension
24. Engage angle selector, flip switch (or press F10)
25. Find max angle flexion (no pelvic restraint rotation)
26. Engage angle selector, flip switch
27. Move pt. to upright position, engage angle selector
28. Explain TDC & have pt. find angle (check twice)
29. Engage angle selector, flip switch
30. level and lock CW at angle of TDC.
31. Move pt. to 0° or greatest angle of extension
32. Engage angle selector, flip switch [act. strain gauge]
33. Zero torso mass with CW adjustment
34. Flip switch to accept
35. Move pt. to upright position, engage angle selector
36. Loosen restraints (release to pt. comfort)
37. Enter gauge reading
38. Enter femur restraint height (patient positioning)
39. Enter seat pad if applicable (patient positioning)
40. Type test remark (PRE FRT)



After tightening lap belt (left photo) and foot board (above), have patient lean to touch toes while you see if pad rotates (photos directly left).

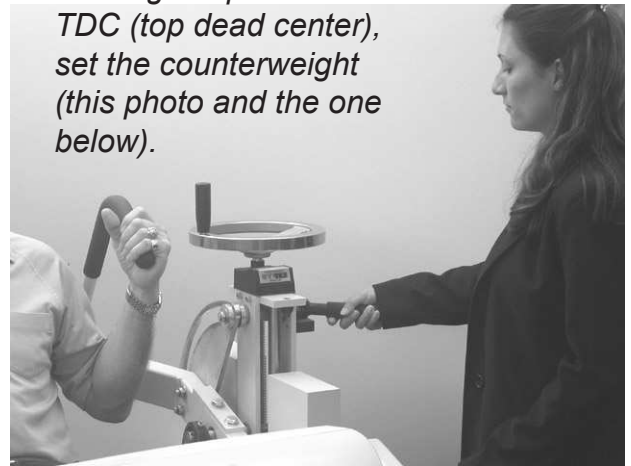


Also have patient attempt to raise his or her heels. If there is much lift, or any rotation in the pelvic pad, continue tightening restraints.

Lumbar Machine Patient Test, Exercise Sessions (cont.)



Position head (occipital lobe) pad, left, and after locating the patient's TDC (top dead center), set the counterweight (this photo and the one below).



41. Explain isometric test: -multiple testing angles
42. 10 sec rest between angles
43. demonst. force application
44. don't push w/head; legs ok
45. build force slowly (3-1-3 s)
46. maintain loose grip
47. exhale during contraction
48. max effort for test accuracy
49. don't push until instructed
50. Ask if pt. understands test instructions
51. Press ENTER to bring up isometric test grid
52. Tighten and check restraints



PRE FRT (ISOMETRIC) TEST

53. Move pt. to full flexion, engage angle selector
54. Have pt. relax, record SE at each angle
55. Force built slowly (3-1-3 s)
56. Exhale during contraction
57. Peak effort reached (contractions not cut short)
58. Encourage / motivate patient
59. Maintain pelvic stabilization (check restraints)
60. 10 sec rest between angles / stretch patient
61. Move patient to upright position
62. Engage angle selector
63. Loosen restraints (release to pt. comfort)
64. Press ENTER to save test



Make sure the patient understands that this system is an effort-dependent measurement. For both valid testing and effective exercise, the patient must give an all-out effort.

Lumbar Machine Patient Test, Exercise Sessions (cont.)

DYNAMIC EXERCISE

65. Determine correct wt. load for DYN (50% max TFT)
66. Set exercise weight on weightstack, tighten chain
67. Enter exercise weight
68. Enter DYN remarks (DYN), bring up test grid
69. Explain DYN: -speed of movement (2-1-4 s)
 70. as many reps as possible
 71. full ROM (listen for tone)
 72. fatigue sensation
 73. exhale during contraction
 74. maintain loose grip
 75. don't push until instructed
76. Ask if pt. understands test instructions
77. Tighten & check restraints
78. Move pt. to full flexion, engage angle selector
79. Release MA lock
80. Flip switch as patient begins to move
81. Controlled speed of movement ≥ 7.0 s per rep)
82. Pt. moves through full ROM (tone)
83. Pt. motivation (last repetition = partial or 140 s limit)
84. Flip switch to end DYN (while pt. in full flexion)
85. Engage MA lock
86. Move patient to upright position
87. Engage angle selector
88. Loosen restraints (release to pt. comfort)
89. Press ENTER to save DYN

POST FRT (ISOMETRIC) TEST

90. Enter test remarks (POST FRT w___, r___, t___)
91. No more than 70 s rest (50 to 70 s)
92. Tighten & check restraints
93. Move pt. to full flexion, engage angle selector
94. Have pt. relax, record SE at each angle
95. Force built slowly (3-1-3 s)

3. Equipment Operation



The angle selector (left hand), movement arm lock (right hand), along with the force/angle switch are located to the patient's right side of machine.



96. Exhale during contraction
97. Peak effort reached (contractions not cut short)
98. Encourage / motivate patient
99. Maintain pelvic stabilization (check restraints)
100. 10 sec rest between angles / stretch patient
101. Move patient to upright position
102. Engage angle selector
103. Loosen restraints, remove pt. from machine
104. Press ENTER to save test

100-POINT CHECKLIST

Cervical Machine Patient Test, Exercise Sessions

PT. PREPARATION

1. Greet pt., explain purpose of visit
2. Have pt. perform static stretches
3. Have pt. void, if necessary
4. Have pt. empty shirt pockets
5. Have pt. remove hair accessories, glasses, etc.

CALIBRATION

6. Move to calibrate, calibration update
7. Counterweight (CW) unlocked
8. Angle selector locked at 90°
9. Loosen chain, remove pins from weightstack
10. Lock gate
11. Pot/SG count #'s within range

[if out of range, contact MedX Tech Support, 800-528-3159]



Angle selector.

TEST SET-UP

12. Select file for test (retrieve or new)
13. Explain: -test sequence (iso/dyn)
 14. need for stabilization (isolation)
 15. should feel pressure from restraints
 16. should not feel pain/numbness
 17. should not feel dizzy, short of breath, etc.
18. Instruct patient to sit upright
19. Place movement arm out of the way
20. Align adam's apple with machine axis (seat ht.)
21. Fasten seat belt & shoulder harness (loosely)
22. Lock gate, (snug torso restraint)
23. Select light wt. (1-2 wt. plates)
24. Tighten chain
25. Have pt. move to full flex., engage angle selector
26. Release MA lock
27. Have pt. perform 5-6 slow reps thru full ROM



Seat height setting.

3. Equipment Operation

Cervical Machine Patient Test, Exercise Sessions (cont.)

28. Adjust seat ht. to eliminate head sliding on pad
29. Have pt. move to full flexion
30. Engage MA lock
31. Move pt. to upright position (MA out of way)
32. Engage angle selector
33. Release gate, tighten harness (secure)
34. Lock gate, tighten torso restraint (secure)
35. Check restraints (shoulder shrug)
36. Find max angle extension
37. Engage angle selector, flip switch (or press F10)
38. Find max angle flexion
39. Engage angle selector, flip switch
40. Locate TDC with pt. looking straight ahead
41. Engage angle selector at pt's TDC, flip switch
42. Level and lock CW at angle of TDC
43. Move patient to 18° (or greatest angle of extension, if more upright)
44. Engage angle selector, flip switch
45. Zero head mass with CW adjustment
46. Flip switch to accept
47. Move pt. to upright position, engage angle selector
48. Loosen torso restraint (release to pt. comfort)
49. Enter gauge reading
50. Enter seat height (patient positioning)
51. Enter seat pad, if applicable (patient positioning)
52. Turn stored energy OFF (test options)
53. Type test remark (Initial IM)
54. Explain 1M test: -multiple testing angles
 55. 10 sec rest between angles
 56. demonstrate force application
 57. apply force w/ back of head
 58. build force slowly (3-1-3 s)
 59. rest hands on gate / cross on lap
 60. exhale during contraction
 61. max effort for test accuracy
 62. don't push until instructed



Once shoulder straps are applied, and gate is closed, see if the patient can elevate (shrug) his or her shoulders (lower photo). If there's any perceptible movement, tighten restraints.



Cervical Machine Patient Test, Exercise Sessions (cont.)

- 63. Ask if pt. understands test instructions
- 64. Press ENTER to bring up isometric test grid
- 65. Tighten and check restraints

ISOMETRIC TEST

- 66. Move pt. to full flexion, engage angle selector
- 67. Force built slowly (3-1-3 s)
- 68. Exhale during contraction
- 69. Peak effort reached (contractions not cut short)
- 70. Encourage / motivate patient
- 71. Maintain torso stabilization (check restraints)
- 72. 10 sec rest between angles / stretch patient
- 73. Move patient to upright position
- 74. Engage angle selector
- 75. Loosen torso restraints (release to pt. comfort)
- 76. Press ENTER to save test

DYNAMIC EXERCISE

- 77. Determine correct wt. load for DYN (80% max TFT)
- 78. Set exercise weight on weightstack, tighten chain
- 79. Enter exercise weight
- 80. Enter DYN remarks (DYN), bring up test grid
- 81. Explain DYN: -speed of movement (2-1-4 s)
 - 82. as many reps as possible
 - 83. full ROM (listen for tone)
 - 84. fatigue sensation
 - 85. exhale during contraction
 - 86. don't push until instructed
- 87. Ask if pt. understands test instructions
- 88. Tighten & check restraints
- 89. Move pt. to full flexion, engage angle selector
- 90. Release MA lock
- 91. Flip switch as patient begins to move
- 92. Controlled speed of movement (≥ 7.0 s per rep)
- 93. Pt. moves through full ROM (tone)



- 94. Pt. motivation (last repetition = partial or 140 s limit)
- 95. Flip switch to end DYN (while pt. in full flexion)
- 96. Engage MA lock
- 97. Move patient to upright position
- 98. Engage angle selector
- 99. Loosen restraints, remove pt. from machine
- 100. Press ENTER to save DYN

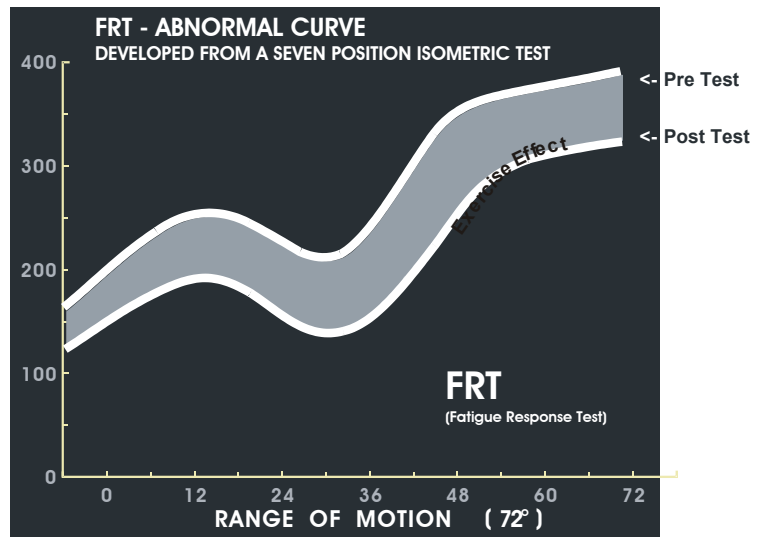
Clinical Fatigue Response Testing

Purpose

The Fatigue Response Test (FRT) is a 3-part test procedure designed to measure the endurance characteristics of a specific muscle group. Information obtained from the FRT can be used to further delineate an exercise prescription based upon the patient's amount of fatigue consequent to the test. It is recommended that the FRT be administered clinically under the following conditions:

- 1) The patient is not limited by joint pain when performing dynamic exercise
- 2) The patient is able to exercise to volitional muscular fatigue
- 3) The patient has demonstrated reliable efforts with previous isometric testing
- 4) The patient has not responded to the recommended standard protocol

Two tests of functional strength. The top of the shaded area shows the level of fresh strength, while the bottom shows remaining strength after the subject was exercised. The shaded area between the two curves is fatigue from the exercise. The dips in the midrange of movement indicate abnormal function. The fact that the same abnormal shape was produced during both tests indicates valid test results.



Procedure

To perform the FRT, a patient is seated and restrained in the MedX machine in order to isolate the target muscle group. The patient then performs a series of maximal effort isometric contractions at multiple joint angles through a pain-free ROM (Pre FRT). After a brief rest, the patient performs as many dynamic variable resistance repetitions as possible using a weightload equal to 50% (lumbar extension) or 80% (cervical extension) of the peak torque from the Pre FRT. Each repetition should be performed through the full painfree ROM in a slow, controlled manner. The patient should perform the concentric portion of the repetition for 2 seconds, pause at full contraction for 1 second, then complete the eccentric portion over a 4 second period (total 7 seconds per repetition). Repetitions should be performed until the patient is unable to move the weightload through a full ROM (volitional fatigue). Immediately following the dynamic repetitions (within 1 minute) the patient performs maximal effort isometric contractions at the same joint angles selected for the Pre FRT (Post FRT). The patient is then released from the machine.

Interpretation

The difference between the Pre FRT and Post FRT isometric tests represents the fatiguing effect of the dynamic exercise. The amount of fatigue (inroad) will vary among individuals, and is indicative of the fiber type characteristics of the lumbar extensor musculature. For example, an inroad $\geq 30\%$ reflects fatigue characteristics of fast twitch muscle fibers, which have a low tolerance to exercise (poor endurance). An inroad $\leq 10\%$ reflects fatigue characteristics of slow twitch muscle fibers, which have a high tolerance to

Clinical Fatigue Response Testing (cont.)

exercise (high endurance). An inroad between 10% and 30% reflects fatigue characteristics of an unknown mixture of fast and slow twitch muscle fibers, with a moderate tolerance to exercise (moderate endurance).

Exercise Prescription

When a motivated individual fails to demonstrate progress (progressive increase in isometric strength, progressive increase in dynamic weightloads) using the standard treatment protocol, his/her exercise prescription may need to be altered based upon the findings from the FRT. Use the following table as a guide:

Percent Fatigue from FRT	Muscle Group	Time Under Load	Repetition Range	Exercise Frequency
≤ 10%	Lumbar/Cervical	105 – 140s	15 - 20	2X/WK
≥ 30%	Lumbar	56 - 84s	8 - 12	1X/WK
	Cervical	70 - 105s	10 - 15	1X/WK

DOs and DON'Ts of Testing

1. **ALWAYS** have one hand on the movement arm and one hand on the angle selector when moving from angle to angle (excluding Cervical Extension machine).
2. **NEVER** disengage the angle selector while in the dynamic mode (when movement arm lock is down).
3. **NEVER** exceed a patient's range of motion.
4. **COMMUNICATE** with the patient before and during testing and training. Explain what they should feel, where they should feel it, and what they should not feel.
5. **REMEMBER** that you are performing an effort-dependent measurement of spinal function. You must get the patient's maximal effort.
6. **MAINTAIN** proper body stabilization throughout the testing/training session.
7. **DON'T** cut an isometric contraction short. Allow enough time for motor unit/muscle fiber recruitment.
8. **SELECT** the proper weightload for training. **CHECK** your selection by getting eye-level to the weight stack.
9. **MOTIVATE** the patient. They will always reach momentary mental failure before reaching momentary muscular failure. The last repetition of a set should be a partial one, performed in a slow, controlled manner.
10. **DON'T** allow the patient to exercise too quickly. Each repetition should take approximately 7 seconds (2 second concentric, 1 second hold, 4 seconds eccentric). The average time per repetition (total exercise time divided by total number of repetitions) should fall between 6.5 and 8.0 seconds per repetition.
11. **RELY** on patient feedback, but do not allow the patient to control the test. This is especially important during the stabilization procedure, and dynamic training.
12. **DON'T** get button/lever happy. When in doubt or confused, **STOP** and **READ THE SCREEN**.

Table 1. Summary of studies testing strength and endurance of the lumbar extensor musculature in LBP

Reference	Participants	Testing	Results	Comments
Trunk Extension Studies				
Kankaanpaa et al. (1998 ^a)	Healthy controls without history of LBP, <i>n</i> = 15	Isometric MVC and isometric endurance to failure at 50%MVC during seated (knees 90°) restrained trunk extension	Significantly lower MVC and time endurance time to exhaustion in CLBP (both <i>p</i> < 0.05)	Those with previous lumbar surgery were excluded
	Middle aged women with CLBP, <i>n</i> = 20			Age, height, body mass and BMI similar between groups
Alaranta et al. (1994)	Never any pain, <i>n</i> = 116	Biering-Sorensen test	Significantly lower endurance time in those with history of LBP (<i>p</i> < 0.05)	
	Pain more than 12 months ago, <i>n</i> = 46			
	Pain during previous 12 month, no disability, <i>n</i> = 166			
	Disabling pain during previous 12 months, <i>n</i> = 147			
McNeil et al. (1980)	Healthy controls, <i>n</i> = 57	Standing trunk extension/flexion MVC with pelvis restrained at top of iliac crest on superior edge of backboard using a belt across the anterior superior iliac spine, and bilateral restraints upon the iliac crests.	Both extension/flexion were lower in CLBP, however extension was reduced to a significantly greater degree shown by significantly lower extension/flexion ratios (<i>p</i> < 0.01)	Participants with sciatica & CLBP had significantly lower extension strength compared to both just CLBP participants and healthy controls (<i>p</i> < 0.01) – with the exception of comparison to females with CLBP (<i>ns</i>)
	CLBP patients, <i>n</i> = 40			
Addison & Schultz (1980)	Healthy controls, <i>n</i> = 57	Standing trunk extension/flexion MVC with pelvis restrained at top of iliac crest on superior edge of backboard using a belt across the anterior superior iliac spine, and bilateral	Both extension/flexion were lower in CLBP, however extension was reduced to a significantly greater degree shown by significantly lower extension/flexion ratios (<i>p</i> < 0.001)	No differences between CLBP and an outpatient CLBP group suggesting common physical deficit despite differences in treatment seeking behaviour
	CLBP patients, <i>n</i> = 33			

		restraints upon the iliac crests.		
Takemasa et al. (1995)	<p>Healthy controls without past history of LBP, $n = 126$</p> <p>CLBP with or without organic lumbar lesions, $n = 123$</p>	Isometric MVC during seated (knees 90°) restrained flexion/extension	Both flexion/extension were significantly lower in CLBP ($p < 0.05$), however extension was reduced to a significantly greater degree shown by significantly higher flexion/extension ratios in lesion group ($p < 0.01$)	<p>No differences CLBP with or without organic lumbar lesions suggesting common physical deficit despite differences symptoms</p> <p>Age, height, body mass and BMI similar between groups</p>
Handa et al. (2000)	<p>Healthy controls without past history of LBP, $n = 60$</p> <p>CLBP patients, $n = 52$</p>	Isometric MVC during seated (knees 90°) restrained flexion/extension	Isometric flexion did not significantly differ between groups, isometric extension was significantly lower in CLBP group ($p < 0.05$)	Age, height, body mass and BMI similar between groups
Suzuki & Endo (1983)	<p>Healthy controls without past history of LBP, $n = 50$</p> <p>CLBP patients with or without root impairment, $n = 90$</p>	Prone trunk extension MVC and flexion with legs both straight and bent at hips and knees with restraint belts across lower extremities	Both straight leg flexion, and trunk extension were significantly weaker in the CLBP group ($p < 0.001$)	Age weight and height similar between groups
Leino et al. (1987)	<p><i>Baseline participants</i></p> <p>Participants with “Good” low back status, $n = 578$</p> <p>Participants with “Intermediate” low back status, $n = 260$</p> <p>Participants with “Bad” low back status, $n = 64$</p> <p><i>Follow-up participants</i></p>	Standing dynamic (baseline) and isometric (follow-up) trunk extension/flexion MVC with buttock and thighs against a supporting plate and ankles tied by a belt	<p>At baseline dynamic flexion was significantly weaker in those with worse low back status ($p < 0.01$) however dynamic extension was significantly weaker only in women ($p < 0.05$)</p> <p>At follow-up isometric flexion was significantly weaker in only men with worse low back status ($p = 0.01$) however isometric extension was significantly weaker in</p>	

	Participants with “Good” low back status, $n = 239$		both men and women ($p < 0.05$)	
	Participants with “Intermediate” low back status, $n = 203$			
	Participants with “Bad” low back status, $n = 210$			
Bayramoglu et al. (2001)	Healthy controls with no history of LBP past 2 years, $n = 20$ CLBP patients, $n = 25$	Standing trunk extension/flexion MVC with stabilised knees and lower back	Both flexion and extension were significantly weaker in CLBP group ($p < 0.05$)	Age and height similar between groups
Nicholaisen & Jorgensen (1985)	(Group 1) LBP that made work impossible, $n = 17$ (Group 2) LBP but not that hindered work, $n = 28$ (Group 3) No history of LBP, $n = 32$	Standing trunk extension/flexion MVC a and isometric extension endurance to exhaustion at 60%MVC with stabilised knees and lower back Biering-Sorensen test	No difference in extension/flexion strength between groups. Isometric endurance significantly lower for Group 1 compared to 2-3 in females and males ($p < 0.05$) Endurance time significantly lower females for Biering-Sorensen test ($p < 0.05$)	Age, weight, height and fat free mass similar between groups except for age being higher in group 1 and weight and fat free mass higher in group 1 for females
Holmstrom et al. (1992)	(Group A) Healthy controls with no history of LBP, $n = 42$ (Group B) CLBP patients with uncertain or negative clinical assessment, $n = 75$ (Group C) CLBP patients with positive clinical assessment, $n = 86$	Standing trunk extension/flexion MVC unrestrained lower extremities Biering-Sorensen test	No difference in extension/flexion strength between groups Extension/flexion ratio was significantly lower in Group C compared to A ($p < 0.05$) Endurance time significantly lower in both Group C and B	Age, weight and height similar between groups

			compared to A for Biering-Sorenson test ($p < 0.01$)	
Salminen et al. (1992)	Healthy children, $n = 38$	Biering-Sorensen test	Both flexion and extension endurance times were significantly lower in LBP groups ($p < 0.05$)	No differences CLBP with or without sciatica suggesting common physical deficit despite differences symptoms
	Children with LBP, $n = 31$	Sit up isometric test with knees at 90^0		
	Children with LBP and sciatica, $n = 7$			
			No difference between LBP and LBP with sciatica was found.	Age, sex, school matched between groups
Hultman et al. (1993)	Healthy controls without history of LBP, $n = 36$	Seated isokinetic/isometric trunk extension/flexion with thighs restrained	All variables, except isokinetic/isometric trunk flexion, were significantly lower in CLBP compared to healthy controls and intermittent LBP patients ($p < 0.05$)	Those with previous lumbar surgery were excluded
	Patients with intermittent LBP, $n = 91$	Biering-Sorensen test		
	CLBP patients, $n = 21$			
Parkkola et al. (1993)	Healthy controls, $n = 60$	Standing isometric trunk extension/flexion MVC with chest, thighs and hips restrained	Extension/flexion MVCs showed a gradient between the three groups from higher to lower.	No statistical data reported
	CLBP patients suitable for active rehabilitation, $n = 38$			
	CLBP patients with serious back problems suitable for moderate rehabilitation only, $n = 10$			
				Incidence of disc degeneration significantly higher in CLBP patients ($p < 0.05$)
				Age, sex, employment and profession matched between groups and BMI similar
Mayer et al. (1989 ^b)	Healthy controls without history of previous LBP, $n = 19$	Isokinetic trunk extension/flexion peak torque unrestrained lower extremities	Both extension and flexion were significantly lower in the postoperative group ($p < 0.05$) with the greatest decrease being in extension strength	There was a significant correlation between trunk extensor strength and muscle density in postoperative patients
	Postoperative spinal disc surgery patients, $n = 46$			
				No information on whether demographic characteristics differed between groups

Crossman et al (2004)	<p>Healthy controls without lasting >3 days in previous 12 months, $n = 32$</p> <p>CLBP patients, $n = 35$</p>	<p>Standing trunk extension/flexion isometric MVC unrestrained lower extremities</p> <p>Biering-Sorensen test</p>	<p>MVC and endurance time significantly lower in CLBP group ($p < 0.05$)</p>	<p>Those with previous lumbar surgery were excluded</p> <p>Age, gender and all anthropometric characteristics similar between groups</p>
Paasuke et al. (2002)	<p>Healthy controls, $n = 12$</p> <p>CLBP patients, $n = 12$</p>	<p>EMG recorded bilaterally from lumbar paraspinal muscles at L3 level 3cm from midline during Biering-Sorensen test to failure</p>	<p>Endurance time was significantly lower in the CLBP group ($p < 0.05$)</p>	<p>Those with previous lumbar surgery were excluded</p> <p>Age and gender matched between participant groups</p> <p>Age, height, body mass and BMI similar among participant groups</p>
Humphrey et al. (2005)	<p>Healthy controls without history of LBP in previous 5 years, $n = 175$</p> <p>CLBP patients, $n = 145$</p> <p>Participants with past history of LBP but no attack within previous 2 years, $n = 30$</p>	<p>Back lift MVC</p>	<p>MVC significantly lower in CLBP patients compared to controls ($p < 0.01$)</p>	<p>Those with previous lumbar surgery were excluded</p> <p>CLBP group was significantly older and had higher body mass and BMI than controls</p>
Suuden et al. (2008)	<p>Healthy controls, $n = 20$</p> <p>CLBP patients, $n = 20$</p>	<p>Biering-Sorensen test</p>	<p>Endurance time significantly lower for CLBP patients compared to controls ($p < 0.05$)</p>	<p>Those with previous lumbar surgery were excluded</p> <p>Age, height and weight and BMI similar between groups</p>
Lariviere et al. (2011)	<p>Healthy controls without history of LBP in previous year, $n = 18$</p> <p>CLBP patients, $n = 18$</p>	<p>Dynamic roman chair trunk extensions to failure</p>	<p>Number of repetitions to failure were significantly less in CLBP patients compared to controls ($p < 0.001$)</p>	<p>Those with previous lumbar surgery were excluded</p> <p>Age, height, weight and BMI similar between groups</p>

Demoulin et al. (2006)	Healthy controls without history of LBP in previous year, $n = 10$ CLBP participants, $n = 10$	Isometric MVC during seated (knees 90°) restrained trunk extension	Extension strength significantly weaker in CLBP ($p < 0.05$)	Those with previous lumbar surgery were excluded
Balague et al. (1993)	Children (10-16yrs) without history of LBP, $n = 79$ Children (10-16yrs) with history of LBP, $n = 38$	Standing isokinetic trunk extension/flexion peak torque unrestrained lower extremities	No significant differences for flexion or extension between groups at any age	
Suter & Lindsay (2001)	Healthy controls, $n = 16$ Golfers with CLBP, $n = 25$	Biering-Sorenson test	No significant difference in endurance time between groups	Age, height and weight similar between groups
Da Silva et al. (2005)	Healthy controls without history of LBP in previous year, $n = 15$ CLBP patients, $n = 13$	Standing trunk extension, prone trunk extension and back lift MVC	No differences between groups	Those with previous lumbar surgery were excluded Age, height and weight similar between groups
Lariviere et al. (2010 ^b)	Healthy controls without LBP lasting 1 wk in previous year, $n = 31$ CLBP patients, $n = 27$	Standing trunk extension/flexion MVC and repetitions to failure (endurance time) with stabilised knees and lower back	No significant difference between health controls and CLBP patients for strength or endurance time Low predicted endurance time was associated with high pain catastrophising in CLBP patients ($p < 0.01$)	Those with previous lumbar surgery were excluded Age, height, weight and BMI similar between groups
Renkawitz et al. (2006)	Healthy tennis players without LBP, $n = 36$ Tennis players with CLBP, $n = 48$	Standing isometric trunk extension MVC with shoulders, pelvis and thighs hips restrained	No association between presence of CLBP and trunk extension strength in either univariate or multivariate logistic regressions	

Isolated Lumbar Extension

Lariviere et al. (2010 ^a)	<p>Healthy controls without LBP lasting 1 wk in previous year, $n = 18$</p> <p>CLBP patients, $n = 18$</p>	Isolated lumbar extension MVC and number of repetition to failure at 60%MVC using customised dynamometer	No significant difference between groups	<p>Those with previous lumbar surgery were excluded</p> <p>Age, body mass, height, BMI, body % and physical activity levels were similar between groups</p>
Cassisi et al. (1993)	<p>Healthy controls without history of LBP, $n = 12$</p> <p>CLBP patients, $n = 21$</p>	Isolated lumbar extension MVC using MEDX	Lumbar extension significantly weaker in CLBP ($p = 0.01$)	<p>13 CLBP patients had undergone previous surgery though no effect upon lumbar extension strength was observed</p> <p>Age and height were similar between groups though body mass was greater in CLBP group</p>
Holmes et al. (1996)	<p>Healthy geriatric female controls, $n = 20$</p> <p>CLBP geriatric female patients, $n = 18$</p>	Isolated lumbar extension MVC using MEDX	Lumbar extension significantly weaker in CLBP ($p < 0.05$)	<p>Age, height and weight similar between groups</p>
Robinson et al. (1992 ^b)	<p>Healthy controls, $n = 12$</p> <p>CLBP patients (53% having had previous surgery), $n = 16$</p>	Isolated lumbar extension MVC using MEDX was performed and 60%MVC determined at full extension for further EMG analysis during isotonic trial (see table 3)	Absolute load used during isotonic trial was significantly lower in the CLBP group compared with the asymptomatic controls ($p < 0.05$)	<p>10 CLBP patients had undergone previous surgery</p> <p>Age, height and weight similar between groups</p>
Nelson et al. (1995)	CLBP patients, $n = 895$	Isolated lumbar extension MVC using MEDX	CLBP baseline data was compared graphically to healthy norms from (Graves et al., 1990 ^a) and shown to considerably weaker.	Patients diagnoses included non-specific CLBP, degenerative disc/arthritis disease, lumbar disc syndrome or spondylolisthesis/spondylolysis

Mooney et al. (1995)	Strip mine workers (90% reported prior LBP), $n = 197$	Isolated lumbar extension MVC using MEDX	Baseline data was compared graphically to healthy norms from (Graves et al., 1990 ^a) and shown to considerably weaker.	
Mooney et al. (1997)	Healthy controls, $n = 8$ CLBP patients, $n = 8$	Isolated lumbar extension MVC using MEDX	CLBP baseline data was compared graphically to both healthy participants in the study and healthy norms from (Graves et al., 1990 ^a) and shown to be considerably weaker.	Patients showed evidence of degenerative disc disease
Boyce et al. (2008)	Small manufacturing plant workers (53% reported LBP), $n = 20$	Isolated lumbar extension MVC using MEDX	Baseline data was compared graphically to healthy norms from (Graves et al., 1990 ^a) and shown to considerably weaker.	

Table 2. Summary of imaging and histochemical studies of the lumbar extensor musculature in LBP

Reference	Participants	Testing	Results	Comments
Imaging Studies				
Hultman et al. (1993)	Healthy controls without history of LBP, $n = 24$	CSA and density of erector spinae using CT at L3 level	Muscle density was significantly lower in CLBP patients compared to both other groups ($p < 0.05$) CSA did not significantly differ between groups	Those with previous lumbar surgery were excluded
	Patients with intermittent LBP, $n = 40$			Age, height, body mass and body composition similar between groups
	CLBP patients, $n = 21$			
Parkkola et al. (1993)	Healthy controls, $n = 60$ CLBP patients suitable for active rehabilitation, $n = 38$	CSA, fat content and grading status graded using 4 classification system of psoas and back muscles (erector	CSA was significantly lower in both CLBP groups compared with controls ($p < 0.001$)	Incidence of disc degeneration significantly higher in CLBP patients ($p < 0.05$)

	CLBP patients with serious back problems suitable for moderate rehabilitation only, $n = 10$	spinae and multifidus) using MRI at L4/L5 level	Back muscle status showed a gradient between the three groups from better to worse. It was significantly worse in severe CLBP patients compared with mild CLBP patients ($p < 0.05$) and healthy controls ($p < 0.001$), and was significantly worse in mild CLBP patients compared with controls also ($p < 0.05$)	Age, sex, employment and profession matched between groups
			Psoas muscles did not differ between groups	
Mayer et al. (1989 ^b)	Healthy controls without history of previous LBP, $n = 19$ Postoperative spinal disc surgery patients, $n = 46$	CSA and muscle density of psoas, erector spinae, rectus abdominus and obliques using CT at L3	Non-significant trends towards reduced CSA in psoas and erector spinae were found in the postoperative group Muscle density of psoas and erector spinae was significantly lower in the postoperative group ($p < 0.001$)	There was a significant correlation between trunk extensor strength and muscle density in postoperative patients No information on whether demographic characteristics differed between groups
Kamaz et al. (2007)	Healthy controls without LBP or leg pain, $n = 34$ CLBP patients, $n = 36$	CSA of total paraspinal, multifidus, quadratus lumborum, psoas and gluteus maximus muscles using CT at L4 upper and lower plates	CSA was significantly reduced in only paraspinal and multifidus at the lower plate in CLBP ($p < 0.01$) CSA was significantly reduced in only multifidus, psoas and quadratus lumborum at the upper plate in CLBP ($p = 0.05$)	Those with previous lumbar surgery were excluded Age and BMI similar in both groups.

			No significant differences between CSA of gluteus maximus	
Sihvonen et al. (1993)	<p>LBP patients who underwent surgery for lumbar spinal stenosis and/or disc herniation 2-6 years prior with good recovery, $n = 14$</p> <p>LBP patients who underwent surgery for lumbar spinal stenosis and/or disc herniation 2-6 years prior regarded as post-operatively failed, $n = 21$</p>	Paraspinal muscle density at L4-L5 level using CT	Muscle density was significantly greater in the group with good recovery compared with the post-operatively failed group ($p < 0.01$)	<p>Lumbar spinal stenosis and/or disc herniation confirmed by CT</p> <p>Age similar between groups</p>
Mooney et al. (1997)	<p>Healthy controls, $n = 8$</p> <p>CLBP patients, $n = 8$</p>	Fatty infiltration and CSA of lumbar paraspinal musculature using MRI from L3 endplate to lower endplate of L5 and graded using 4 classification system	<p>CLBP patients showed evidence of fatty infiltration compared with controls 5/8 showing severe</p> <p>All patients showed greater fatty infiltration of paraspinal muscles compared with any other lumbar muscles</p> <p>No difference in CSA between groups</p>	<p>No statistical data reported</p> <p>Patients showed evidence of degenerative disc disease</p>
Hides et al. (1994)	<p>Healthy controls, $n = 51$</p> <p>First episode acute LBP patients, $n = 26$</p>	CSA of multifidus on left and right sides using real-time ultrasound at L2, L3, L4, L5 and S1	Asymmetry was significantly greater corresponding to level of symptoms in LBP patients compared with normal participant between-side differences ($p < 0.001$)	Only comparisons of between side differences were reported between LBP patients and normal participants. Manual extraction of data on CSA from figure 2 in ref [96] suggests that average CSA

				of asymptomatic side in LBP patients did not differ significantly from healthy participant's largest side.
				Age, height and weight similar between groups
Mannion et al. (2000)	CLBP patients, $n = 59$	CSA of erector spinae, quadratus lumborum and psoas using MRI at L3/L4 and L4/L5 levels	CSA showed association with lean body mass and age, but no association with symptom duration	No healthy control group for comparisons Those with previous lumbar surgery were excluded
Mengiardi et al. (2006)	Healthy controls without history of LBP in previous 2 years, $n = 25$ CLBP patients, $n = 25$	CSA of multifidus and longissimus fat content and semi-quantitative grading using 5 classification system using MRI at L4-L5 level	CLBP patients showed significantly greater fat content in the multifidus ($p < 0.05$) No difference found using semi-quantitative system	Those with previous lumbar surgery were excluded Age, sex and BMI matched between participant groups
Cooper et al. (1992)	Recent onset LBP patients (symptoms less than 18 months), $n = 43$ CLBP patients (symptoms more than 18 months), $n = 44$	CSA of paraspinal and psoas muscles using CT at L4 normalised to L4 bone CSA	Normalised paraspinal and psoas CSAs significantly reduced in CLBP compared to recent onset group ($p < 0.05$)	All participants technically chronic as defined by Frymoyer [108] Lumbar surgery in preceding 18 months were excluded, though most CLBP patients included ($n = 31$) had undergone prior surgery CLBP participants also significantly older
Bouche et al. (2011)	Post-discectomy patients pain free, $n = 18$	Muscle CSA and fat CSA of total paraspinal, erector spinae, multifidus and psoas+iliac muscle using CT	Muscle CSA of erector spinae and multifidus significantly smaller in pain patients ($p < 0.05$)	Level of operation was not found to be a significant factor and so suggests a general deconditioning of the

	Post-discectomy patients with LBP, $n = 18$	at L3, L4, and L5 normalised to L3 bone CSA	Fat CSA significantly greater in psoas of pain patients ($p < 0.05$)	lumbar musculature independent of surgery Age and BMI similar between groups
Danneels et al. (2000)	Healthy controls without history of previous LBP, $n = 23$ CLBP patients, $n = 32$	Total CSA and muscle CSA of total paraspinal, erector spinae, multifidus and psoas muscles using CT at upper L3, and upper and lower L4 normalised	Total CSA of paraspinal and multifidus muscles significantly smaller at lower L4 in CLBP ($p < 0.05$) No significant difference for erector spinae or psoas	Those with previous lumbar surgery were excluded in addition to those who had participated in training for the lower back muscles in the previous 3 months Age, height, weight and activity similar between groups
Alaranta et al. (1993)	CLBP patients, $n = 39$	Fat content of lumbar paraspinal musculature using CT at three lowest levels and 4 level classification system	Fat content was moderately positively associated with disability score on Oswestry index ($p < 0.05$) but not with age, sex, body mass, BMI, degree of disc degeneration, or facet joint osteoarthritis	Those with previous spinal fusion surgery were excluded however 16 patients had undergone previous surgery for lumbar disc herniation >1 year prior.
Kader et al. (2000)	CLBP patients, $n = 75$	Atrophy of the multifidus compared with normal results from [106] using MRI and 3 level classification system	80% of participants showed moderate to severe multifidus atrophy	Those with previous lumbar surgery were excluded Significant association between multifidus atrophy and leg pain ($p < 0.01$)
Barker et al. (2004)	CLBP patients with unilateral pain, $n = 50$	CSA of left and right multifidus and psoas muscles using MRI at level of symptoms and one level above and below	CSA of both multifidus and psoas significantly smaller on symptomatic side at all levels ($p < 0.05$)	Those with previous lumbar surgery were excluded Multifidus atrophy consistently relatively greater than psoas atrophy at all levels

				Significant association between psoas atrophy and pain, nerve root compression and symptom duration.
				Significant association between multifidus atrophy and symptom duration
Kjaer et al. (2007)	<p>Adults aged 40 years, $n = 409$ (85% reporting LBP ever, 70% reporting LBP in previous year)</p> <p>Adolescents aged 13 years, $n = 439$ (41% reporting LBP ever, 22% reporting LBP in previous year)</p>	Fat content of multifidus using MRI at 3 lower lumbar levels using 3 level classification system	<p>Association between fat content of multifidus for LBP ever (Odds Ratio = 7.2) and LBP in previous year (Odds Ratio = 3.6) in adults.</p> <p>No association between fat content of multifidus in adolescents</p>	Associations increased when controlling for effect moderators including gender, BMI, physical workload, leisure and sports activities.
Hyun et al. (2007)	<p>Healthy controls without lumbosacral radiculopathy or disc herniation, $n = 19$</p> <p>LBP patients with unilateral lumbosacral radiculopathy, $n = 14$</p> <p>LBP patients with disc herniation but no lumbosacral radiculopathy, $n = 25$</p>	Total CSA and muscle CSA of multifidus using MRI at L3/L4, L4/L5, and L5/S1	<p>Total CSA, muscle CSA and ratio of the two were significantly reduced in both LBP groups involved sides compared to controls at most levels ($p < 0.05$) and ratio at L3/L4 ($p < 0.05$)</p> <p>No difference between LBP groups for total CSA, muscle CSA and ratio of the two</p> <p>Ratio of involved side CSA to uninvolved side CSA was significantly different in radiculopathy patients compared to both controls and the other LBP patients ($p < 0.01$ to 0.05)</p>	Those with previous lumbar surgery were excluded from LBP control group

Kalichman et al. (2010)	<p>Healthy controls without LBP in previous year, $n = 150$</p> <p>Patients who had suffered from LBP of at least 1 month within previous year, $n = 37$</p>	Density of erector spinae and multifidus muscles using CT at L3, L4, and L5	<p>Muscle density was not associated with LBP</p> <p>Reduced muscle density was significantly associated with presence of facet joint osteoarthritis, spondylolisthesis and disc narrowing ($p < 0.05$)</p> <p>Both non-adjusted and adjusted means for muscle density showed significant associations with the presence and severity of LBP for the paraspinal muscles ($p \leq 0.0001$), and lateral abdominals ($p < 0.05$).</p>	
Hicks et al. (2005)	<p>Controls aged 70-79 years without LBP in previous year, $n = 861$</p> <p>Patients with mild LBP in previous 12 months, $n = 244$</p> <p>Patients with moderate LBP in previous 12 months, $n = 299$</p> <p>Patients with severe/extreme LBP in previous 12 months, $n = 111$</p>	Total CSA and density of paraspinal, and lateral abdominal muscles using CT at L4-L5 level	<p>Both non-adjusted and adjusted means for muscle density showed significant associations with the presence and severity of LBP for the paraspinal muscles ($p \leq 0.0001$), and lateral abdominals ($p < 0.05$).</p>	
Kang et al. (2007)	<p>CLBP patients with lumbar degenerative kyphosis undergoing corrective surgery, $n = 54$</p> <p>CLBP control patients, $n = 54$</p>	CSA and muscle to disc CSA ratio of psoas, erector spinae and multifidus was assessed at L4/L5 level and fatty infiltration of psoas, erector spinae and multifidus assessed at L3/L4 using three grade classification using MRI	<p>CSA and muscle to disc CSA ratios for all muscles were significantly lower in the lumbar degenerative kyphosis group compared with controls ($p < 0.001$) with regression analysis showing multifidus wasting to be most strongly associated ($p < 0.001$)</p> <p>Severe fatty infiltration was significantly more common in lumbar degenerative</p>	<p>No healthy control group for comparisons</p> <p>Those with previous lumbar surgery were excluded from CLBP control group</p> <p>Age and sex matched between groups and symptom durations were similar</p> <p>Body mass and BMI was significantly higher in CLBP controls</p>

kyphosis compared to CLBP controls ($p < 0.05$)

No difference in degenerative changes (degenerative disc disease, herniation's, stenosis or spondylolithesis) between groups

Histochemical Studies

Crossman et al (2004)

Healthy controls without LBP lasting >3 days in previous 12 months, $n = 32$

CLBP patients, $n = 35$

Percutaneous biopsy of paraspinal muscle (specific location not noted) for fibre CSAs and fibre typing.

No significant differences between groups for any fibre histochemical comparisons

Those with previous lumbar surgery were excluded

Age, gender and all anthropometric characteristics similar between groups

Weber et al. (1997)

LBP patients undergoing posterior surgery, $n = 61$ (posterior surgery for persistent pain Op1 $n = 43$, posterior surgery for removal of internal fixation Op2 $n = 32$)

Biopsy of multifidus at L3, L4, L5 or S1 level for fibre diameter, fibre typing and pathological changes

Pathological changes were common in biopsy specimens from Op1

No healthy control group for comparisons

Type II atrophy was associated with age and severity of pain in biopsy specimens from Op1

Muscular alterations were present in patients undergoing Op1 however surgery may have caused further alterations as presence of changes were increased in Op2

Patients undergoing Op2 showed significantly greater pathological changes compared with biopsy specimens from Op 1 ($p = 0.05$)

Biopsy specimens were taken from 14 patients at the same level in both Op1 and Op2 with 70% of normal

Rantanen et al (1993)	Patients from ref [123] who underwent surgery for lumbar disc herniation 5 years prior, <i>n</i> = 18	Biopsy of multifidus taken 1cm laterally from spinous process of the level immediately below the previously herniated disc (L4/L5 and/or L5/S1) for fibre narrow diameter, fibre typing, atrophy/hypertrophy and pathological changes	<p>biopsies at Op1 showing alterations at Op2</p> <p>No changes in fibre type distribution, atrophy/hypertrophy factors were noted compared with baseline</p> <p>Type I fibre size significantly increases</p>	<p>Level of herniation and thus biopsy did not influence results</p> <p>Patients with both 'positive' and 'negative' outcomes from original surgery were compared showing decreased pathological changes in 'positive' group compared with their persistence in 'negative' group</p>
Sihvonen et al. (1993)	<p>LBP patients who underwent surgery for lumbar spinal stenosis and/or disc herniation 2-6 years prior with good recovery, <i>n</i> = 14</p> <p>LBP patients who underwent surgery for lumbar spinal stenosis and/or disc herniation 2-6 years prior regarded as post-operatively failed, <i>n</i> = 21</p>	Biopsy of paraspinal muscle taken from site of abnormal myelogram finding for fibre atrophy	Local denervation atrophy observed in all but one post operatively failed patients	<p>Lumbar spinal stenosis and/or disc herniation confirmed as absent by CT</p> <p>Age similar between groups</p> <p>No statistical data reported</p>
Mannion et al. (2000)	CLBP patients, <i>n</i> = 59	Biopsy of belly of lateral tract of left erector spinae at L3/L4 level for fibre CSA, fibre typing and pathological changes	<p>Symptom duration was a strong predictor of both fibre type changes towards a more type IIx phenotype</p> <p>Pathological changes were common and significantly associated with age and showed a trend to</p>	<p>No healthy control group for comparisons</p> <p>Those with previous lumbar surgery were excluded</p>

			association with symptom duration	
Ford et al. (1983)	Patients undergoing surgery for lumbar disc herniation reporting LBP duration between 3 and 52 weeks, $n = 18$	Biopsy of erector spinae (sacrospinalis) 1cm lateral to tip of spinous process and multifidus 1cm from inferior border of lamina at L5 level for fibre typing, fibre narrow diameter and pathological changes	No differences between left and right sides Pathological changes were common but varied and not impacted by side of herniation	No healthy control group for comparisons Side of herniation did not affect results
Zhu et al. (1989)	Patients undergoing surgery for lumbar disc herniation, $n = 22$	Biopsy of erector spinae from side and level of herniation 1cm lateral to top of spinous process for fibre typing, atrophy/hypertrophy and pathological changes	Proportion of fibres types for type I, type IIa and type IIb were 68%, 10.6% and 21.4% respectively Type II atrophy was common with type IIb most frequent and severe 18 patients showed evidence of pathological changes	No healthy control group for comparisons
Mannion et al. (1997 ^a)	Healthy controls without history of LBP requiring time of work or doctors attention, $n = 29$ CLBP patients undergoing posterior surgery, $n = 31$ (First time operation $n = 22$, patients undergoing second operation $n = 9$)	Biopsy of belly of lateral tract of left erector spinae at L3 level for fibre narrow diameter, fibre typing and pathological changes	Smaller proportion of type I and greater proportion of type IIb fibres as both % and % fibre type area were found in CLBP patients compared to healthy controls ($p < 0.05$) Pathological changes did not differ between groups	Age, sex and body mass matched between participant groups
Fidler et al. (1975)	Patients with LBP, $n = 17$ Cadavers within 24 hours of death, $n = 3$	Biopsy of multifidus from separated muscle cut transversely, taken during operation	Grouping of slow fibres appeared in addition to reduced CSA of fast fibres in LBP	No details on nature of operation No statistical data reported

Mattila et al (1986)	<p>Patients undergoing first time surgery for lumbar disc herniation, $n = 41$</p> <p>Control participants without history of LBP undergoing autopsy within 48 hours of death, $n = 12$</p>	<p>Biopsy of multifidus taken during operation or autopsy at L4/L5 and L5/S1 levels for fibre narrow diameter, fibre typing, atrophy/hypertrophy and pathological changes</p>	<p>Relative numbers of type I and type II fibres did not correlate with age nor differ significantly between groups</p> <p>Pathological changes were significantly more frequent in patients compared to controls ($p < 0.01$)</p>	<p>Biopsy taken from deltoid to rule out systemic congenital myopathy</p> <p>Level of herniation and thus biopsy did not influence results</p>
Zhao et al. (2000)	<p>LBP patients undergoing first time surgery for lumbar disc herniation, $n = 19$</p>	<p>Biopsy of multifidus taken during operation from transversospinal corner on both left and right sides at the level of herniation (L4/L5 or L5/S1) for fibre CSA, fibre narrow diameter, fibre typing and pathological changes</p>	<p>CSAs and diameters of both type I and type II fibres were significantly smaller on the side of herniation ($p < 0.05$)</p> <p>Strength factor (%fibre type x fibre CSA) of type II fibres was also lower on side of herniation ($p < 0.05$)</p> <p>Pathological changes were present in both sides but more severe on the side of herniation</p>	<p>No healthy control group for comparisons</p> <p>Location of pain symptoms was associated with muscle alterations</p>
Bajek et al. (2000)	<p>Patients undergoing surgery for lumbar disc herniation, $n = 76$</p> <p>Control participants without history of neuromuscular disease undergoing autopsy within 48 hours of sudden death, $n = 41$</p>	<p>Biopsy of multifidus on side of herniation and at level of herniation in patients (L3/L4, L4/L5, or L5/S1) and L4/L5 level in controls 1cm lateral from midline deeper than the aponeurosis of erector spinae for fibre typing and fibre diameter</p>	<p>Greater proportion of type I and smaller proportion of type IIa type IIb fibres in patients compared with controls in males only ($p < 0.05$)</p> <p>Fibre diameter in type I fibres was significantly greater in patients compared to controls ($p < 0.05$) and for type IIa and type IIb was significantly</p>	<p>Age was similar between groups</p>

			greater than controls for males only ($p < 0.05$)	
Yoshihara et al. (2001)	LBP patients undergoing first time surgery for lumbar disc herniation, $n = 29$	Biopsy of multifidus taken during operation immediately after start of surgery dissected from L4 and L5 muscle bands on both sides for fibre typing, fibre size and pathological changes	<p>Fibre size of type 2 fibres was significantly smaller than type I at all biopsy sites</p> <p>Fibre size did not differ between sides at L4 for type I or type II fibres but fibre size was significantly smaller at L5 on side of herniation for both type I and type II fibres ($p < 0.01$)</p> <p>No difference in fibre type proportions</p> <p>Pathological changes were present at all biopsy sites but only significantly different between sides, with greater frequency on side of herniation at L5</p>	<p>No healthy control group for comparisons</p> <p>No difference between level of biopsy</p>

Table 3. Summary of studies testing fatigability with EMG of the lumbar extensor musculature in LBP

Reference	Participants	Testing	Results	Comments
Kankaanpaa et al. (1998 ^a)	<p>Healthy controls without history of LBP, $n = 15$</p> <p>Middle aged women with CLBP, $n = 20$</p>	EMG recorded bilaterally from gluteus muscles and lumbar paraspinal muscles at L3/L4 and L5/S1 levels 2cm laterally from midline of spinous process during isometric MVC and isometric endurance to failure at 50%MVC during seated (knees 90°) restrained trunk extension	Neither EMG amplitude or fatigue indices data differed between groups for the paraspinal muscles	<p>Those with previous lumbar surgery were excluded</p> <p>Age, height, body mass and BMI similar between groups</p>

Lariviere et al. (2010 ^a)	<p>Healthy controls without LBP lasting 1 wk in previous year, $n = 18$</p> <p>CLBP patients, $n = 18$</p>	EMG recorded bilaterally from gluteus maximus, biceps femoris and vastus medialis muscles and lumbar paraspinal muscles at L4, L3, L1, and T10 levels during isolated lumbar extension MVC and repetitions to failure at 60%MVC using customised dynamometer	None of the EMG fatigue indices data differed between groups for the paraspinal muscles	<p>Those with previous lumbar surgery were excluded</p> <p>Age, body mass, height, BMI, body % and physical activity levels were similar between groups</p>
Crossman et al (2004)	<p>Healthy controls without lasting >3 days in previous 12 months, $n = 32$</p> <p>CLBP patients, $n = 35$</p>	EMG recorded bilaterally from lumbar paraspinal muscles at L4-L5 level during standing isometric trunk extension for 60 seconds at 60%MVC and during the Biering-Sorensen test	EMG fatigue indices were similar between groups for the Biering-Sorensen test and also the 60%MVC test	<p>Those with previous lumbar surgery were excluded</p> <p>Age, gender and all anthropometric characteristics similar between groups</p>
Paasuke et al. (2002)	<p>Healthy controls without history of LBP or LBP in previous year, $n = 12$</p> <p>CLBP patients, $n = 12$</p>	EMG recorded bilaterally from lumbar paraspinal muscles at L3 level 3cm from midline during Biering-Sorensen test to failure	EMG indices of fatigue showed significantly greater fatigue in the CLBP group compared to controls ($p < 0.05$)	<p>Those with previous lumbar surgery were excluded</p> <p>Age and gender matched between participant groups</p> <p>Age, height, body mass and BMI were similar between participant groups</p>
Humphrey et al. (2005)	<p>Healthy controls without history of LBP in previous 5 years, $n = 175$</p> <p>CLBP patients, $n = 145$</p> <p>Participants with past history of LBP but no attack within previous 2 years, $n = 30$</p>	EMG recorded bilaterally from lumbar paraspinal muscles at L4/L5 during a back lift test with 66.66%MVC for 30 seconds	<p>EMG indices of fatigue showed significantly greater fatigue in the CLBP compared to controls ($p < 0.05$)</p> <p>Logistic regression showed high sensitivity (0.65) and specificity (0.75) in classifying CLBP patients</p>	<p>Those with previous lumbar surgery were excluded</p> <p>CLBP group was significantly older and had higher body mass and BMI than controls</p>

			Past history participants could not be adequately discriminated from either group	
Suuden et al. (2008)	Healthy controls, $n = 20$ CLBP patients, $n = 20$	EMG recorded bilaterally from lumbar paraspinal muscles at L3 3 cm from midline during Biering-Sorenson test to failure	No significant differences in EMG indices of fatigue between groups	Those with previous lumbar surgery were excluded Age, height and weight and BMI similar between groups
Lariviere et al. (2011)	Healthy controls without history of LBP in previous year, $n = 18$ CLBP patients, $n = 18$	EMG recorded bilaterally from gluteus maximus, biceps femoris and lumbar paraspinal muscles at L4, L3, L1, and T10 levels during dynamic roman chair trunk extensions to failure	No significant differences in EMG indices of fatigue between groups	Those with previous lumbar surgery were excluded Age, height, weight and BMI similar between groups
Suter & Lindsay (2001)	Healthy controls without history of LBP, $n = 16$ Golfers with CLBP, $n = 25$	EMG recorded bilaterally from lumbar paraspinal muscles at T12 and L4-L5 level 3cm from midline during Biering-Sorenson test to failure	No significant difference in EMG fatigue indices between groups	Age, height and weight similar between groups
Da Silva et al. (2005)	Healthy controls without history of LBP in previous year, $n = 15$ CLBP patients, $n = 13$	EMG recorded bilaterally from lumbar paraspinal muscles at T10, L1, L3, and L5 levels during standing trunk extension and back lift at 50%MVC for 60 seconds, and during Biering-Sorenson test for 60 seconds	No difference in EMG fatigue indices between groups	Those with previous lumbar surgery were excluded Age, height and weight similar between groups
Lariviere et al. (2010 ^b)	Healthy controls without LBP lasting 1 wk in previous year, $n = 31$	EMG recorded bilaterally from gluteus maximus, biceps femoris and lumbar paraspinal muscles at L5, L3,	EMG indices of fatigue showed significantly greater fatigue in CLBP patients with high catastrophising	Those with previous lumbar surgery were excluded

	CLBP patients, $n = 27$	L1, and T10 levels during standing trunk extension/flexion MVC and repetitions to failure (endurance time) with stabilised knees and lower back	compared with CLBP patients with low catastrophising ($p < 0.01$)	Age, height, weight and BMI similar between groups
Robinson et al. (1992 ^b)	Healthy controls never treated for LBP and without LBP in previous year, $n = 12$ CLBP patients (53% having had previous surgery), $n = 16$	EMG recorded bilaterally from lumbar paraspinal muscles at L1-L2 level during isolated lumbar extension at 60%MVC in full extension for 12-13 repetitions	EMG amplitude in millivolts decreased across repetitions in asymptomatic participants compared with a significantly flatter curve in the CLBP group ($p < 0.05$)	Age, height and weight similar between groups
Roy et al. (1989)	Healthy controls, $n = 12$ CLBP patients, $n = 12$	EMG recorded bilaterally from lumbar paraspinal muscles at L1, L2 and L5 levels during standing isometric trunk extension for 60 seconds at 40%MVC, 60%MVC and 80%MVC	Discriminant analysis of EMG fatigue indices successfully classified 92% controls, 82% CLBP at 40%MVC, 67% controls, 75% CLBP at 60%MVC and 84% controls, 91% CLBP at 80% MVC	Those with previous lumbar surgery were excluded Age, height and weight similar between groups
Roy et al. (1995)	Healthy controls without history of LBP, $n = 42$ CLBP patients (43% having had previous surgery), $n = 28$	EMG recorded bilaterally from lumbar paraspinal muscles at L1, L2 and L5 levels during standing isometric trunk extension for 30 seconds at 40%MVC and 80%MVC	Discriminant analysis of EMG fatigue indices successfully classified 85% CLBP patients and 86% healthy controls	CLBP patients heterogeneous with respect to symptoms and history (75% had disc herniation and 43% had undergone previous surgery)
Mayer et al. (1989 ^a)	Healthy controls, $n = 11$ CLBP patients, $n = 10$	EMG recorded bilaterally from lumbar paraspinal muscles at L3 level 3cm from midline during 10 isometric trunk extension holds on a roman chair lasting 15 seconds each and	EMG indices of fatigue showed significantly greater fatigue in the CLBP group compared to controls ($p < 0.01$)	Those with previous lumbar surgery at level of EMG placement were excluded Age and torso weight similar between groups

		with 10 seconds rest between each hold		
Peach & McGill (1998)	<p>Healthy controls without history of LBP in previous 2 years, $n = 18$</p> <p>CLBP patients, $n = 21$</p>	<p>EMG recorded from lumbar paraspinal muscles at T9 level 5cm from midline, L3 level 3cm from midline, and L5 level 1-2cm from midline respectively during semi-standing isometric trunk extension for 30 seconds at 60%MVC and then after a 60 second rest during a further 10 second extension at 60%MVC</p>	<p>EMG indices of fatigue showed significantly greater fatigue in the CLBP compared to controls ($p < 0.05$)</p> <p>Discriminant analysis of EMG fatigue indices successfully classified 100% controls and 93.75% CLBP patients</p> <p>Logistic regression was equally powerful using two parameters with concordance of 92.4%</p>	<p>Age, height and weight similar between groups</p>
Roy et al. (1990)	<p>Varsity rowers without LBP, $n = 17$</p> <p>Varsity rowers with LBP in past year, $n = 6$</p>	<p>EMG recorded bilaterally from lumbar paraspinal muscles at L1, L2 and L5 levels during standing isometric trunk extension for 30 seconds at 80%MVC and then after a 60 second rest during a further 5 second extension at 80%MVC</p>	<p>Discriminant analysis of EMG fatigue indices successfully classified 93% controls and 100% LBP participants</p>	<p>Age, height and weight similar between groups</p>
Biedermann et al. (1991)	<p>Healthy controls without history of LBP, $n = 22$</p> <p>CLBP patients, $n = 27$</p>	<p>EMG recorded bilaterally from lumbar paraspinal muscles at L2-L3 and L4-L5 levels during standing with a 11.6 pound dumbbell held in outstretched arms for 45 seconds followed by a 5 minute recovery and the repetition of the 45 second</p>	<p>CLBP patients were classified into 'avoiders' or 'confronters'</p> <p>Discriminant analysis of EMG fatigue indices successfully classified 88.9% 'avoiders', 66.7% 'confronters' and 59.1% controls</p>	<p>Age, height, weight and arm length similar between groups</p> <p>Continuum of fatigue seen between avoiders>confronters>controls, however pain duration differed significantly between avoiders and confronters (8.57 ± 6.22)</p>

		trial – all adjusted for arm length differences		years and 1.60±0.76 years respectively)
Klein et al. (1991)	Varsity rowers without LBP, <i>n</i> = 17 Varsity rowers with LBP in past year, <i>n</i> = 8	EMG recorded bilaterally from lumbar paraspinal muscles at L1, L2 and L5 levels during standing isometric trunk extension for 30 seconds at 80%MVC and then further 10 second extensions at 80%MVC at 1 minute, 2 minutes, 5 minutes, 10 minutes and 15 minutes into recovery	Discriminant analysis of EMG fatigue indices showed most successful classification at 1 and 2 minute recovery, classifying for 1 and 2 minutes respectively 88% and 100% of LBP participants and 100% and 88% of controls	Age, height and weight similar between groups
Mannion et al. (1997 ^b)	Healthy controls without history of LBP, <i>n</i> = 10 LBP patients, <i>n</i> = 12	EMG recorded bilaterally from lumbar paraspinal muscles at T10 and L3 level 3-4cm from midline during Biering-Sorenson test for 60 seconds	MFS was greater in LBP group indicating greater fatigue but just failed to achieve significance (<i>p</i> = 0.10)	Age, height and weight similar between groups Mean values for MFS were similar to those in prospective study which did achieve significance in predicting first time LBP

Table 4. Summary of prospective studies of lumbar extensor musculature deconditioning in LBP

Reference	Participants	Testing	Results	Comments
Biering-Sorenson (1984)	Men aged between 30, 40, 50, and 60 years old, <i>n</i> = 449 Women aged between 30, 40, 50, and 60 years old	Biering-Sorensen test conducted at baseline 1 year follow-up with questionnaire concerning first time occurrence, recurrence or persistence of LBP	First time occurrence was significantly associated with low endurance time	
Leino et al. (1987)	<i>Baseline participants</i> Participants with “Good” low back status, <i>n</i> = 578	Standing dynamic trunk extension/flexion maximum repetitions performed over 30 seconds with buttock and	Trunk strength was not predictive of low back symptoms or status at follow up.	

Luoto et al. (1995)	Participants with "Intermediate" low back status, $n = 260$	thighs against a supporting plate and ankles tied by a belt conducted at baseline	
	Participants with "Bad" low back status, $n = 64$	Standing isometric trunk extension/flexion MVC with buttock and thighs against a supporting plate and ankles tied by a belt conducted at	
	<i>Follow-up participants</i>	10 year follow-up in addition to questionnaire and assessment of low back symptoms and status	
	Participants with "Good" low back status, $n = 239$		
	Participants with "Intermediate" low back status, $n = 203$		
	Participants with "Bad" low back status, $n = 210$		
Luoto et al. (1995)	Healthy participants without history of LBP in previous year at baseline, $n = 167$	Biering-Sorensen test and questionnaire regarding previous and present LBP conducted at baseline	Endurance time was significantly associated with first time occurrence of LBP when adjusted for age, sex and occupation ($p < 0.05$)
		75% of participants were available for follow-up at 1 year with the same questionnaire, $n = 126$	Endurance time broken into tertiles (poor, medium, good) showed a non- linear dose-response relationship with first time occurrence of LBP ($p < 0.04$)
			Relative odds ratio compared to 'good' for 'medium' and 'poor' were 1.4 (95% CI 0.4 - 4.2) and 3.4 (95% CI 1.2 – 10.0) respectively

Gibbons et al. (1997)	Healthy participants without history of LBP in previous year at baseline, $n = 43$	<p>Isokinetic back lift MVC, psychophysical back lift test, Biering-Sorensen test, CSA, proton-density weighted signal, and T2-weighted signal of erector spinae, quadratus lumborum, psoas major and total paraspinal muscle using MRI, and interview regarding previous and present LBP conducted at baseline</p> <p>Interviews regarding LBP were conducted at 1 year follow-up</p>	<p>Neither back lift, psychophysical back lift or endurance time differed between those with and without LBP at follow-up, nor were they associated with frequency of LBP at follow-up</p> <p>Neither CSA, proton-density weighted signal, or T2-weighted signal differed between those with and without LBP at follow-up, however, total paraspinal CSA, and proton-density weighted signal and T2-weighted signal of erector spinae, quadratus lumborum, psoas major were significantly associated with frequency of LBP at follow-up ($p < 0.05$)</p>
Mannion et al. (1997 ^b)	Healthy nurses without history of LBP, $n = 200$	<p>EMG recorded bilaterally from lumbar paraspinal muscles at T10 and L3 level 3-4cm from midline during Biering-Sorenson test and maintenance of 80%MVC for 28 seconds at baseline</p> <p>Postal questionnaire regarding LBP conducted at 1 year follow-up</p>	<p>13% developed serious first time LBP during the follow-up period</p> <p>EMG indices of fatigue during Biering-Sorenson showed greater fatigue was significantly associated with development of first time LBP at follow-up ($p < 0.05$) however endurance time was not associated with first time LBP</p>

Rissanen et al. (2002)	Participants from the Mini-Finland Health Survey, $n = 535$	Dynamic trunk extension/flexion maximum repetitions performed over 30 seconds with buttock and thighs against a supporting plate and ankles tied by a belt conducted at baseline Average 12 year follow-up to time until retirement due to work disablement, death or end of observation period for primary diagnosis as cause of work disability	At follow-up of 56 incident cases 15 were due to back disorders Adjusted relative risks in multiple models showed trunk extension performance significantly predicted back disorder disability risk ($p = 0.04 - 0.002$)	
Newton et al. (1993)	Healthy participants without history of LBP, $n = 70$	Isokinetic trunk extension, flexion, rotation, and back lift MVC and psychophysical lift conducted at baseline 1 year follow-up with questionnaire concerning first time occurrence, recurrence or persistence of LBP	23% developed LBP during the follow-up period, yet at least 6 months after initial assessment in all cases None of the isokinetic measures differed between those who did and those who did not develop LBP	Those with previous lumbar surgery were excluded
Reimer et al. (1994)	Healthy prospective order selector employees for 1989, $n = 122$ Healthy prospective order selector employees for 1990, $n = 122$ Healthy prospective order selector employees for 1991, $n = 122$	Dynamic lift capacity, isokinetic trunk extension, flexion, rotation, and back lift MVC and psychophysical lift conducted at baseline to determine placement in employment as an order selector in a warehouse grocery distributor 2 year follow-up with questionnaire concerning first time occurrence,	After implementation of prospective evaluation for employment placement in 1989, incidence of low back injuries were significantly reduced by 32% in 1990 and 41% in 1991 ($p < 0.001$)	

		recurrence or persistence of LBP		
Batt'ie et al. (1989)	Employees working for a large aircraft manufacturer (n = 497 reporting LBP in previous 10 years), n = 2178	Isometric MVC for torso, arm and leg lift was conducted at baseline 4 year follow-up conducted for claims related to low back injuries or LBP	Participants with higher MVC for arm, leg and torso lift were at higher risk for LBP and low back injury ($p = 0.01$, 0.03 , and 0.26 respectively). When adjusted for age and sex however no association was present.	Due to an injury rate of 0.6% during torso lift testing it was discontinued. n = 495 participants completed torso lift testing, n = 2158 completed arm lift testing, and n = 2102 completed leg lift testing
Lee et al. (1999)	Healthy student participants without history of LBP, n = 67	Isokinetic trunk extension, flexion, and rotation MVC conducted at baseline. 5 year follow-up concerning LBP incidence	27% developed first time LBP during the follow-up period Ratio of extension/flexion strength at baseline was significantly lower in participants who developed first time LBP, ($p < 0.05$)	Age, height, weight and smoking habits similar between groups
Kujala et al. (1996)	Healthy participants without history of LBP, n = 262	Standing isometric trunk extension/flexion MVC was conducted at baseline 5 year follow-up with questionnaire was conducted regarding type, frequency, severity and functional limitations of LBP	47% developed first time LBP during the follow-up period, 11% of these reporting it as being of monthly frequency, 17% reporting radiating limb pain, and 2% having been hospitalised due to LBP Trunk extension/flexion was not associated with development of first time LBP	Age, weight and BMI similar between groups Height, occupational physical demands, and occupational musculoskeletal loading was significantly associated with first time LBP ($p < 0.05$)

Chaffin et al. (1978)	Pre-employed plant workers in a variety of jobs involving manual lifting, $n = 551$	<p>Isometric MVC for torso, arm and leg lift in addition to job specific demands was conducted at baseline</p> <p>Preventative effectiveness of strength relative to job demands were evaluated by examining incidence and severity of low back injuries over an 18 month follow-up period</p> <p>Participants were grouped into tertiles relating to their individual strength relative to their job demands</p>	As job strength requirements exceeded participant strength the incidence and severity of low back injuries increased at a ratio of 3:1 across the tertiles	
Keyserling et al. (1980)	Pre-employed plant workers applying for a range of 20 varied jobs, $n = 71$	<p>Isometric MVC for torso and arm lift, and push in/out in addition to job specific demands was conducted at baseline</p> <p>Preventative effectiveness of strength relative to job demands evaluated by placing of experimental ($n = 20$) group into jobs matching strength whereas control group ($n = 51$) were not</p> <p>Incidence of musculoskeletal injuries were evaluated over a 1 year follow-up period</p>	During the follow-up period the control group experienced 19 incidences of musculoskeletal injuries compared to 0 in the experimental group	Age, weight and height similar between groups
Salminen et al. (1974)	<p>Healthy children, $n = 38$</p> <p>Children with LBP, $n = 31$</p>	Biering-Sorensen test, sit up isometric test with knees at	Both flexion and extension endurance times were significantly lower in LBP	Age, sex, school matched between groups

	Children with LBP and sciatica, $n = 7$	90° and MRI conducted at baseline 3 year follow-up period evaluating LBP ever, LBP in past 12 months, and recurrent/continuous LBP	groups ($p < 0.05$) at baseline and follow-up yet endurance time was not predictive of development of first time LBP
Sjolie & Ljunggren (2001)	Healthy adolescents, $n = 86$	Biering-Sorensen test and questionnaire regarding LBP conducted at baseline 3 year follow-up period with questionnaire was completed	High mobility /endurance time ratios were significantly associated with development of LBP at follow-up when adjusted for gender, LBP at baseline, and well-being and physical activity at follow-up (OR 1.5 - 1.9, 95% CI 1.1 – 3.2, $p < 0.05$)
Adams et al. (1999)	Healthy nurses without history of LBP, $n = 262$ Healthy nurses who had previously suffered with 'non-serious' LBP, $n = 141$	Biering-Sorensen tests, isometric back lift MVC and back lift at 80%MVC for 20 seconds while EMG recorded from T10 and L3 conducted at baseline 3 year follow-up (every 6 months) conducted using questionnaire regarding LBP in previous 6 months	Endurance time at 3 year follow-up was significantly associated with development of serious LBP ($p < 0.01$) and approached significance for any LBP ($p < 0.058$) Neither back lift nor indices of fatigue were associated with development of LBP
Mostardi et al. (1992)	Healthy nurses without history of LBP, $n = 171$	Isokinetic back lift MVC conducted at baseline Injury reports used to examine incidence of low back injury over 2 years follow-up	9% sustained low back injuries during the follow-up period There was no significant difference in strength at baseline between those who reported low back injury

			during follow-up and those who did not	
Cady et al. (1979)	Healthy fire-fighters without LBP, $n = 1652$	<p>Isometric back lift MVC conducted at baseline</p> <p>Incidence of prior low back injuries examined subsequent to baseline measurements – no specific follow-up duration was noted</p> <p>Participants were split into percentiles for 'Most Fit' (84-100 percentile), 'Middle Fit' (17-83 percentile) and 'Least Fit' (0-16 percentile)</p>	7.14% sustained low back injuries in the 'Least Fit' group, 3.19% sustained low back injuries in the 'Middle Fit' group, and 0.77% sustained low back injuries in the 'Most Fit' group	Mean age increased with decreasing fitness levels between the three groups
Mooney et al. (1996)	Workers without history of LBP in a ship-building firm in the 3 highest Physical Demand Characteristic categories across 32 jobs, $n = 152$	<p>Isolated lumbar extension MVC using MEDX</p> <p>2 year follow-up of low back injury and LBP claims</p>	<p>9% sustained low back injuries during the follow-up period the majority occurring in the heavy PDC category (64%)</p> <p>Isolated lumbar extension strength was not predictive of low back injuries and only 2 of those participants injured had below normal strength</p>	<p>Age, height and weight was similar amongst PDC categories and in those injured and uninjured</p> <p>Low back injury rates were significantly higher in heavy and very heavy PDC categories ($p < 0.0001$)</p>
Stevenson et al. (2001)	<p>Spinning operators from DuPont without history of LBP, $n = 72$</p> <p>Spinning operators from DuPont suffering from LBP in previous 2 years, $n = 46$</p>	<p>EMG recorded bilaterally from lumbar paraspinal muscles at T10 and L3 level 3-4cm from midline during Biering-Sorenson test</p> <p>2 year follow-up period at 6 month intervals for LBP</p>	EMG indices of fatigues entered final model and were significantly predictive of LBP ($p = 0.035$)	Other factors in final predictive model included age, peak thoracic acceleration, leg strength/endurance, however psychosocial factors were largely absent.

	<p>Spinning operators from DuPont suffering from LBP in previous year, $n = 31$</p>	<p>experiences in previous 6 months</p>	
<p>Heydari et al. (2010)</p>	<p>Healthy participants classified as either 'No History of LBP', 'CLBP' or 'Past History of LBP', $n = 105$</p>	<p>EMG recorded bilaterally from lumbar paraspinal muscles at L4/L5 level during back lift test maintaining 2/3MVC for 30 seconds at baseline and follow-up</p> <p>2 year follow-up participants were asked to classify themselves as 'worse', 'better, or 'the same'</p>	<p>At follow-up 76 classified themselves as 'the same', 13 'better' and 16 'worse'</p> <p>EMG indices of fatigue showed greater fatigue was significantly associated with development of first time LBP and with self-classification at follow-up ($p < 0.05$)</p>

7.7 Determining the reliability of a custom built seated stadiometry set-up for measuring spinal height in participants with chronic low back pain

Abstract

Indirect measurement of disc hydration can be obtained through measures of spinal height using stadiometry. However, specialised stadiometers for this are often custom-built and expensive. Generic wall-mounted stadiometers alternatively are common in clinics and laboratories. This study examined the reliability of a custom set-up utilising a wall-mounted stadiometer for measurement of spinal height using custom built wall mounted postural rods. Thirteen participants with non-specific chronic low back pain (CLBP) underwent measurement of spinal height on three separate consecutive days at the same time of day where 10 measurements were taken at 20 second intervals. Intra-individual absolute standard error of measurement (SEM) was calculated for spinal height using the first of the 10 measures, the average of 10 measures, and the shrinkage across the 10 measures. SEMs were 3.1mm, 14.2mm and 2.6mm respectively. SEMs for first of the 10 measures and the shrinkage across the 10 measures suggests this custom set-up for measuring spinal height changes is suitable use as an outcome measure in either research or clinical practice in participants with CLBP.